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MEDICAL

Neurology

Dr. Conrad Fischer

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Chapter 1: Diseases of the Spinal Cord

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Spinal Cord Compression

Spinal Cord Compression — Etiology

- This is a neurologic emergency!
- Secondary to
  - Cancer
  - Epidural abscess
  - Hematoma

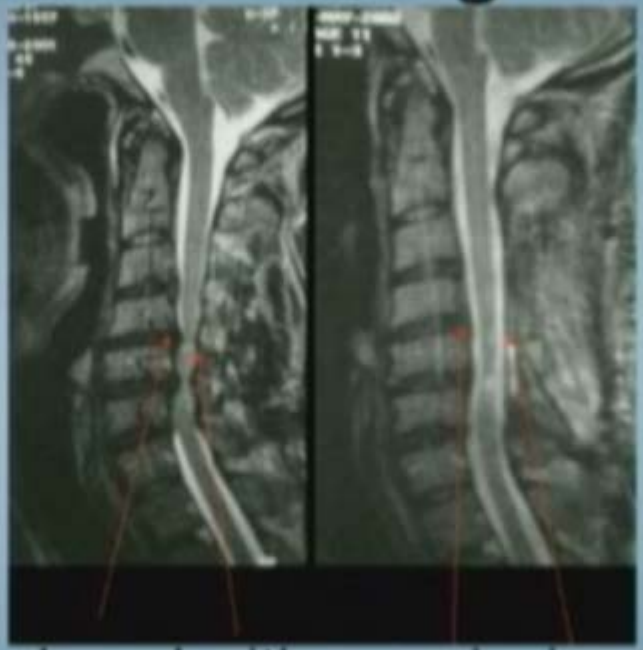
Spinal Cord Compression — Presentation

- Insidious
  - Sensory disturbance
  - Lower extremity weakness
  - Sphincter dysfunction
  - Pain
  - Hyperreflexia

Spinal Cord Compression — Treatment

- High dose dexamethasone
- Further treatment differs with etiology
  - Tumors
    - Radiotherapy
  - Herniation, abscess, or hematoma
    - Surgical decompression

Spinal Cord Compression: Before and After Neurosurgical Repair



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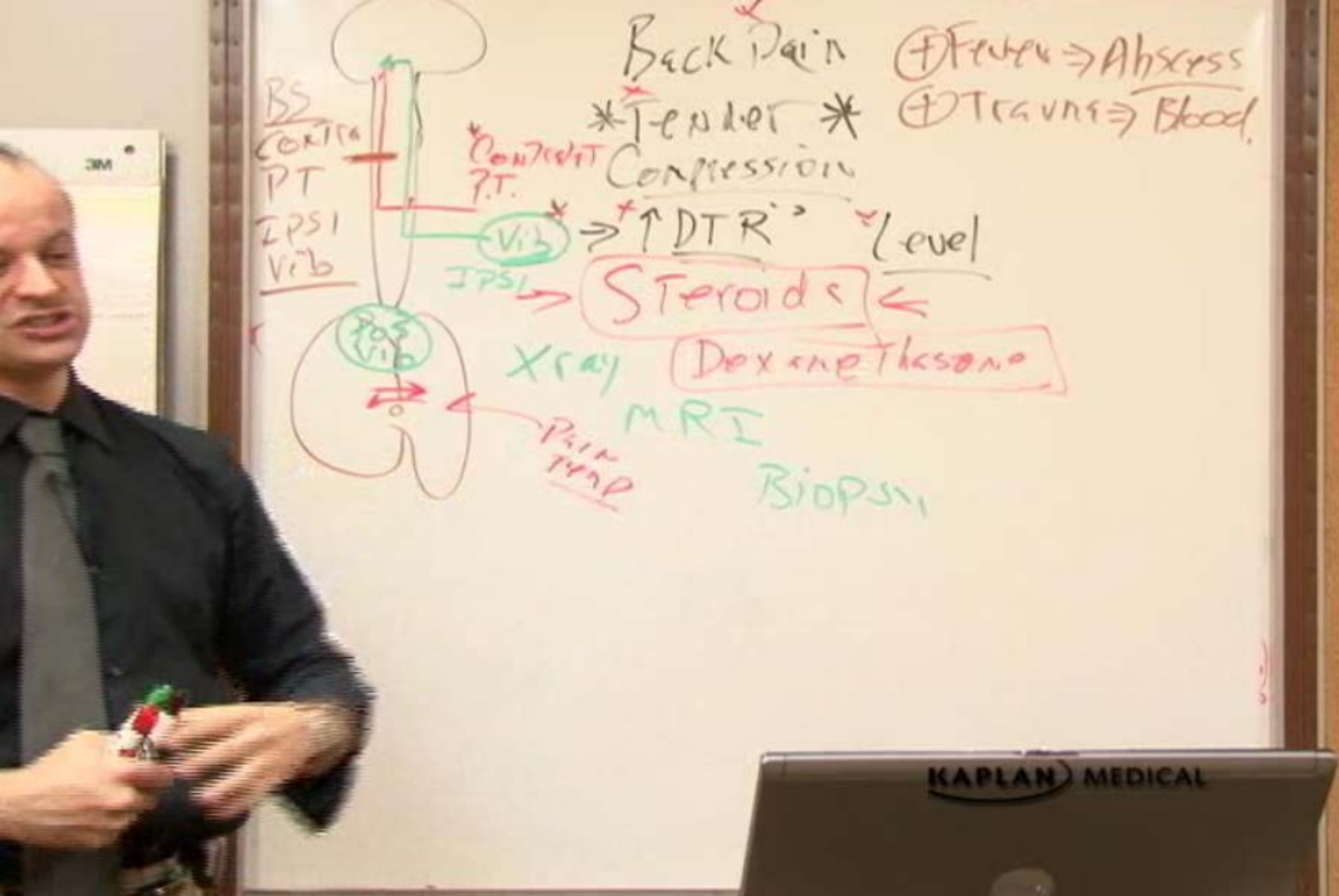
Back Pain
(+) Fever ⇒ Abscess
(+) Trauma ⇒ Blood
* Tender *
Compression
↑ DTR's
Level
BS
Contra
PT
Ipsi
Vib
Contralat
P.T.
Vib
Ipsi
Steroids
Dexamethasone
X-ray
MRI
Biopsy
Pain
Temp
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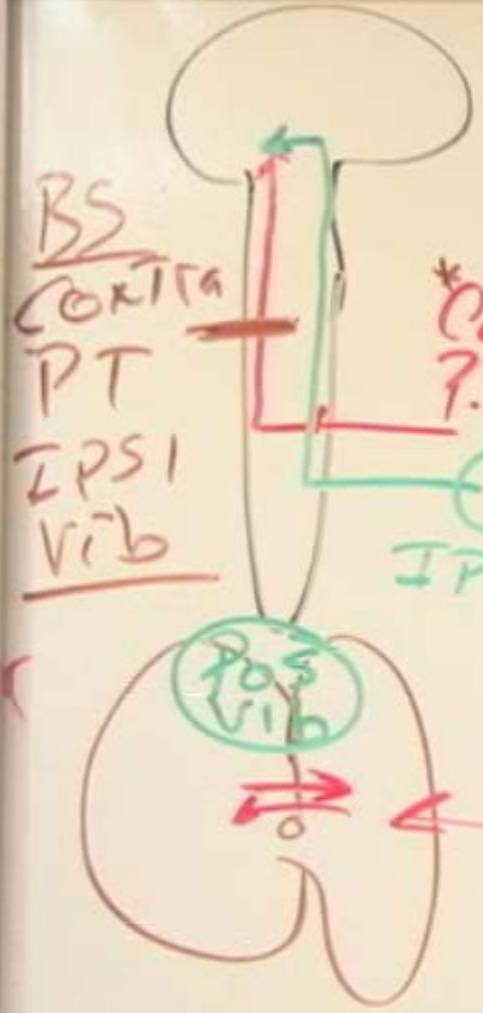


Back Pain

Tender

Compression

⇒ ↑ DTR's

Level

Steroids

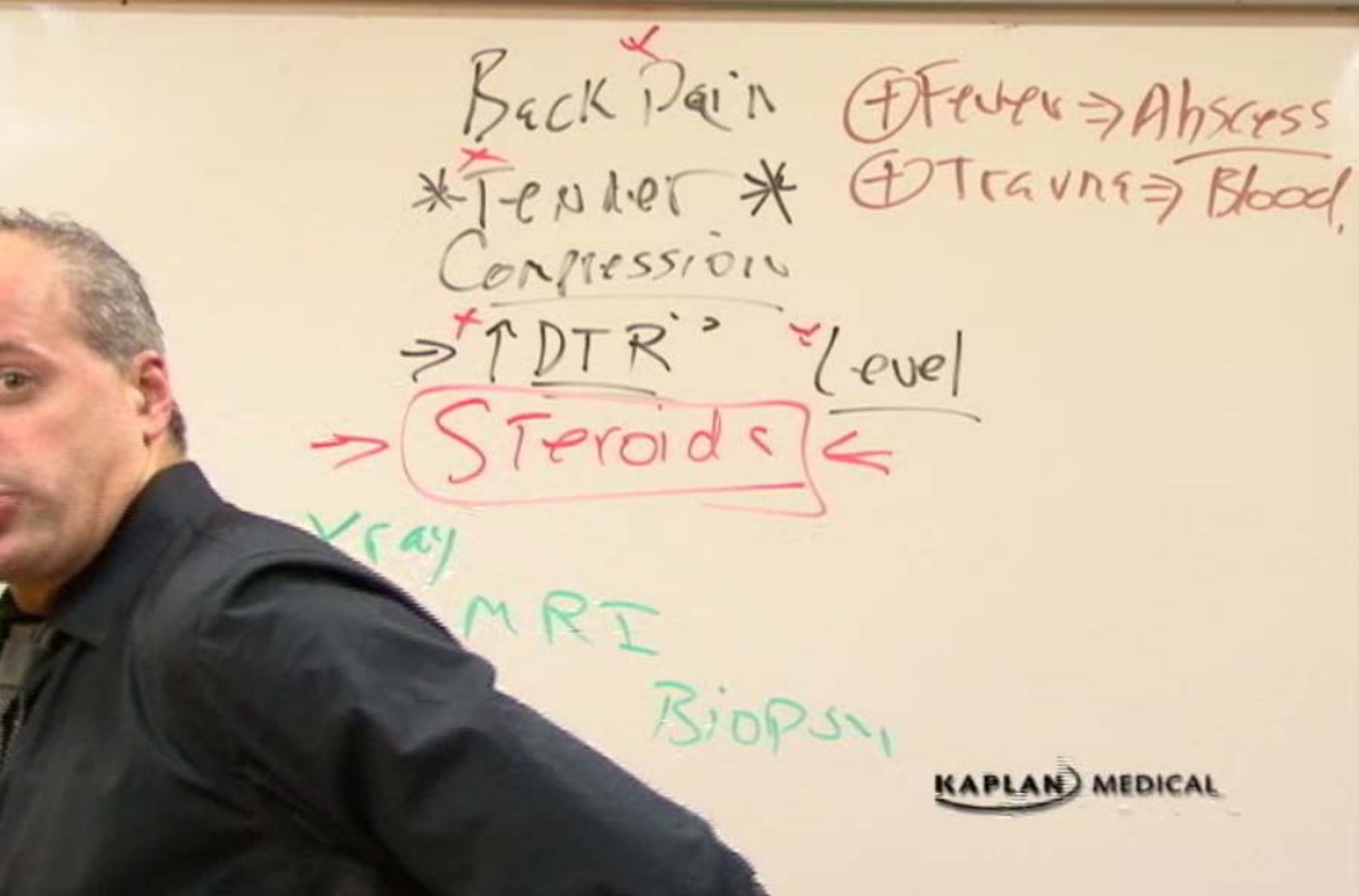
X ray

MRI

Bone Scan

(+) Fever ⇒

(+) Trauma


Back Pain
+Fever ⇒ Abscess
Tender
+Trauma ⇒ Blood
Compression
⇒ ↑ DTR's
Level
Steroids
X-ray
MRI
Biopsy
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Spinal Cord Compression

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Syringomyelia

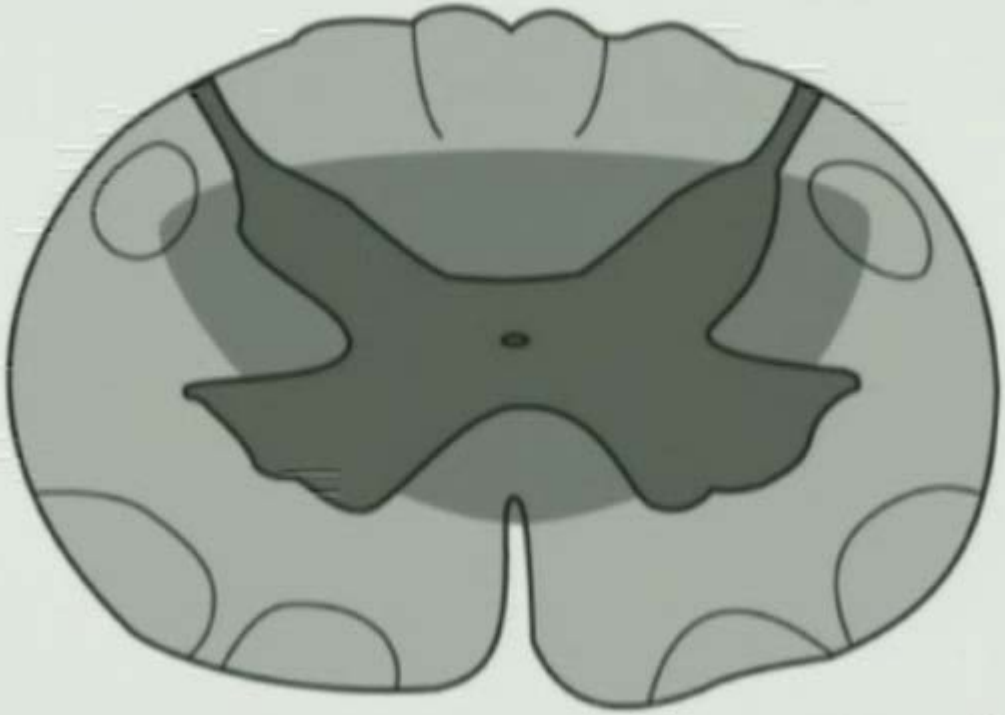
Syringomyelia — Etiology

- Most common in the cervical spinal cord
- Communicating
  - Arnold-Chiari Malformation
- Non-Communicating
  - Trauma
  - Tumors

Syringomyelia — Presentation

- Cape-like loss of pain, temperature and sensation to light touch across the neck and arms
- Sparing of tactile sensation, position and vibration
- Absent reflexes

Syringomyelia



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Syringomyelia — Treatment

- Surgical

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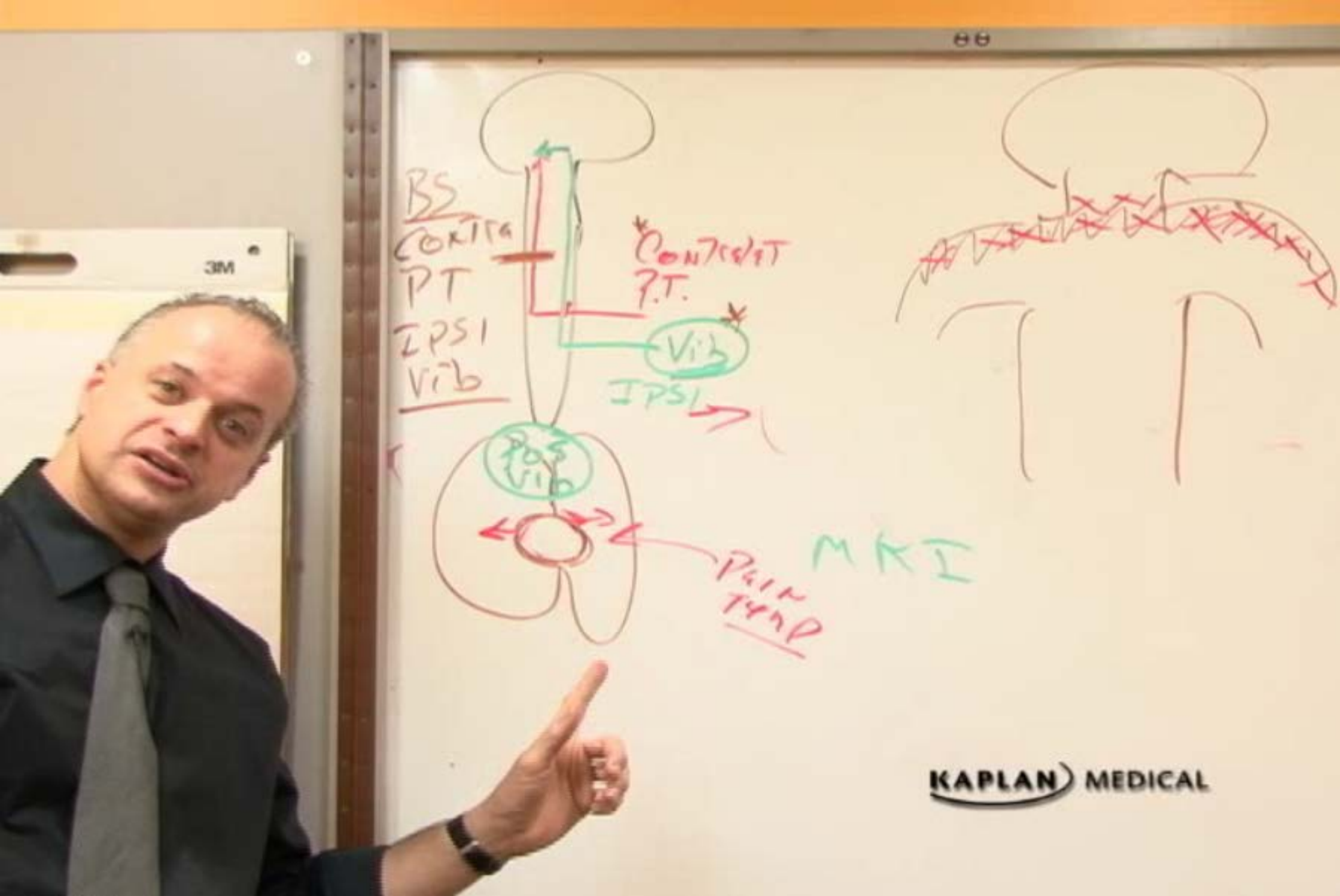
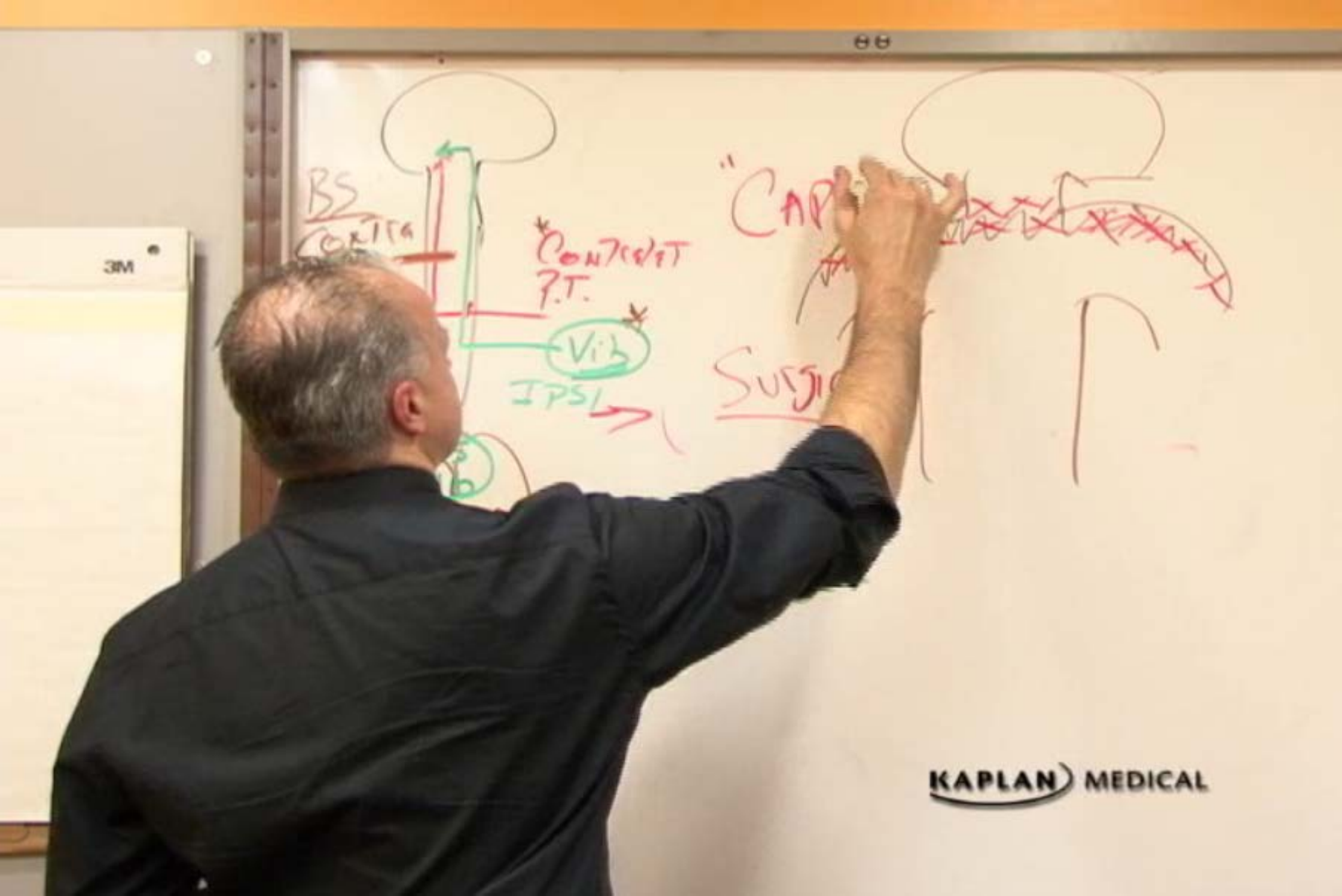

BS
CONTRA
PT
IPSI
Vib
CONTRAST
P.T.
Vib
IPSI
MRI
KAPLAN MEDICAL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Syringomyelia

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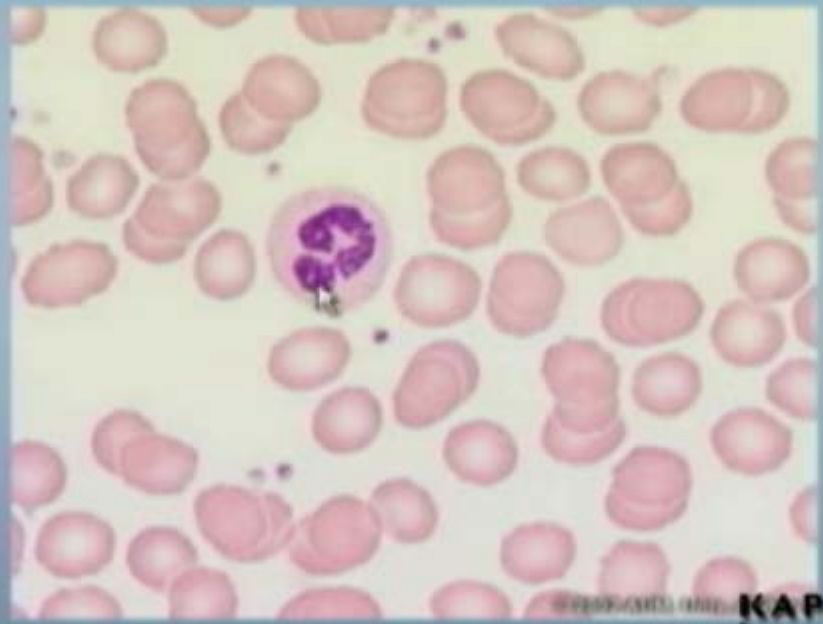
Subacute Combined Degeneration

Subacute Combined Degeneration — Etiology

- Secondary to B-12 deficiency

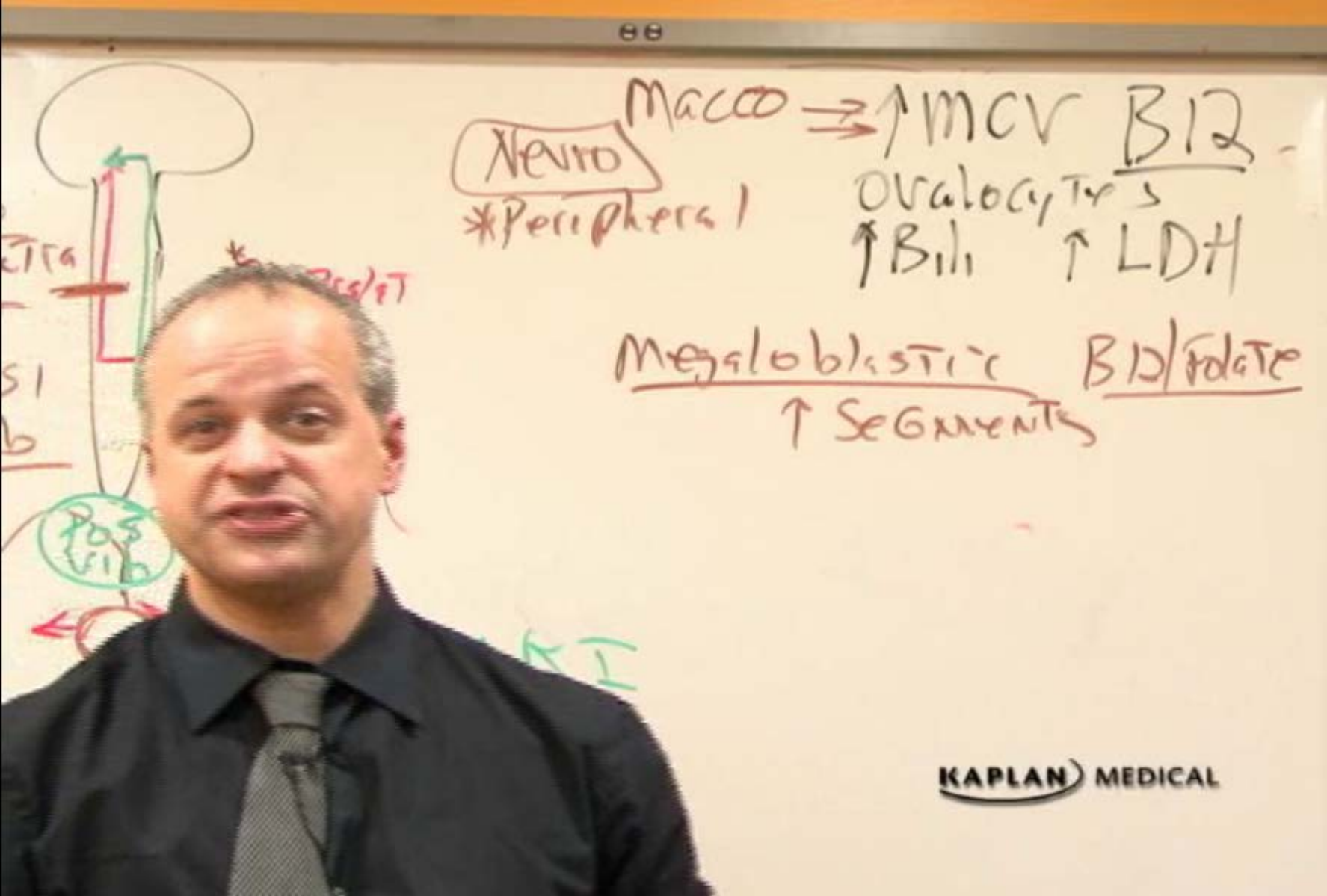


Subacute Combined Degeneration — Clinical Presentation

- History:
  - Distal paresthesias and extremity weakness
  - Progresses to spastic paresis and ataxia if left untreated
- Physical Exam
  - Combined deficit of vibration and position sense
  - Pyramidal signs

Subacute Combined Degeneration — Diagnosis and Treatment

- Diagnosis
  - Low serum B-12
  - May have macrocytic anemia with hypersegmented neutrophils
- Treatment
  - B-12 replacement


Macro ⇒ ↑MCV B12
Neuro
*Peripheral
Ovalocytes
↑Bili ↑LDH
Megaloblastic B12/Folate
↑ Segments
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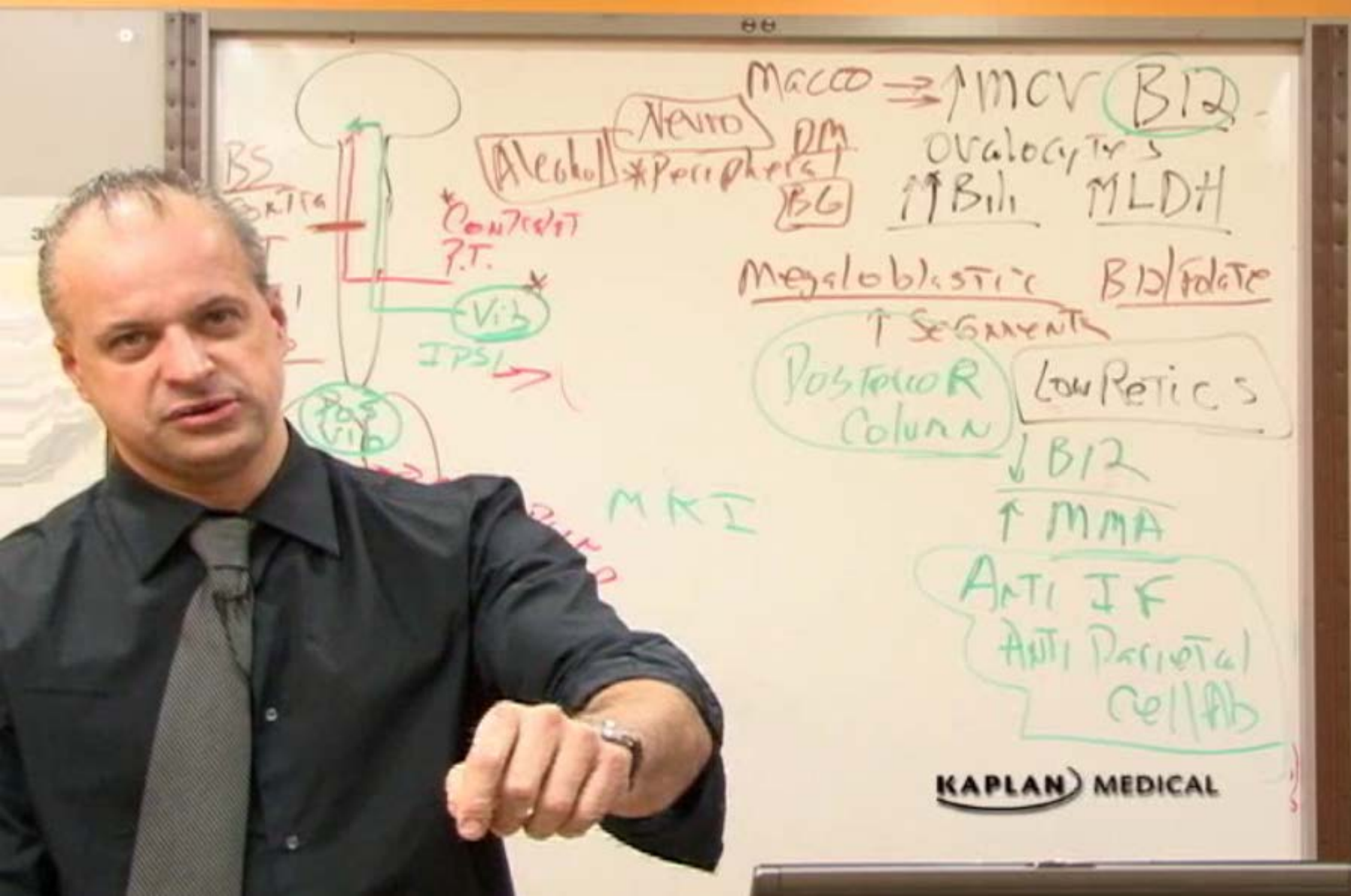
MacCO ⇒ ↑MCV B12
Alcohol Neuro *Peripheral
DM
BG
Ovalocytes
↑↑Bili MLDH
Megaloblastic B12/Folate
↑ Segments
Contrast
P.T.
Vib
IPSI
BS
MRI
Posterior Column
Low Retics
↓ B12
↑ MMA
Anti IF
Anti Parietal Cell Ab
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↑ SEGMENTS

POSTERIOR COLUMN

LOW RETICS

↓ B12

↑ MMA

ANTI IF

ANTI PARIETAL CELL Ab

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Subacute Combined Dengeneration

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Anterior Spinal Artery Occlusion

Anterior Spinal Artery Infarction — Presentation

Acute onset of flaccid paralysis

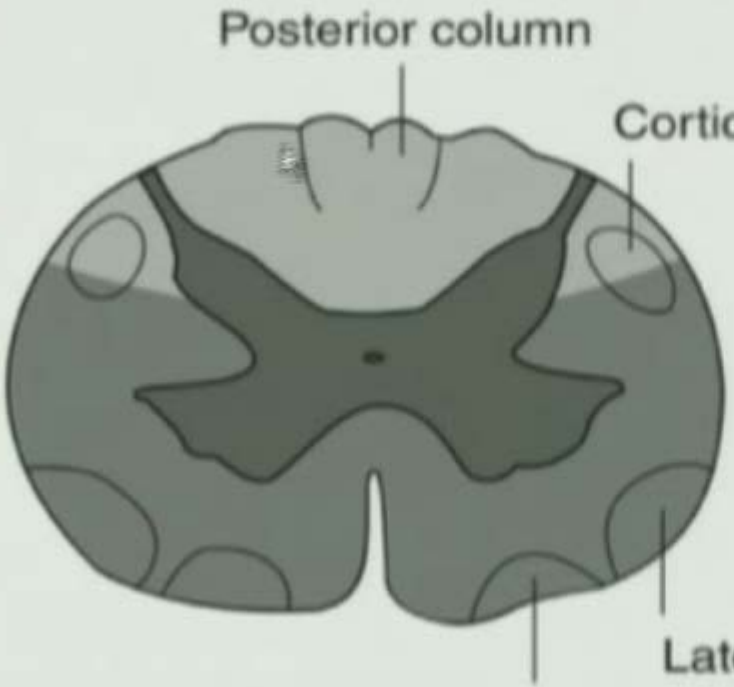
Days to weeks later

Spastic paresis

Anterior Spinal Artery Infarction — Presentation

- Everything is lost except position and vibratory senses
  - Pain and temperature- Lateral spinothalamic tracts
  - Motor- Anterior columns
  - Autonomic- Intermediolateral columns

Anterior Spinal Artery Infarction



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Anterior Spinal Artery Infarction — Treatment

- Supportive

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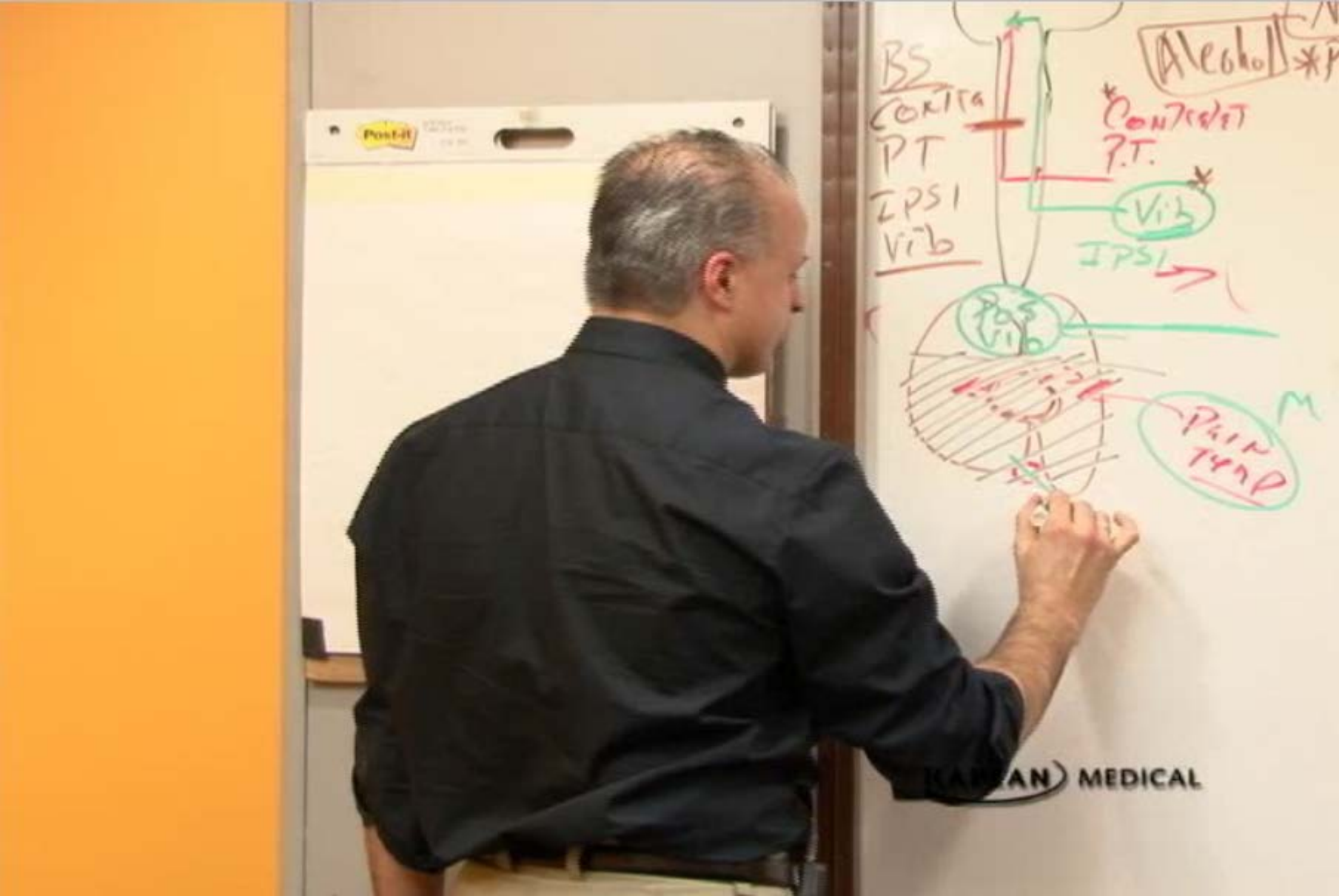
BS
CONTRA
PT
IPSI
Vib
Alcohol
CONTRA
P.T.
Vib
IPSI
Pain
Temp
MEDICAL


Post-it
3M
BS
CORTEX
PT
IPSI
Vib
Alcohol
CONTRAST
P.T.
Vib
IPSI
MRI
PAIN TEMP
MOTOR
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Anterior Spinal Artery Infarction

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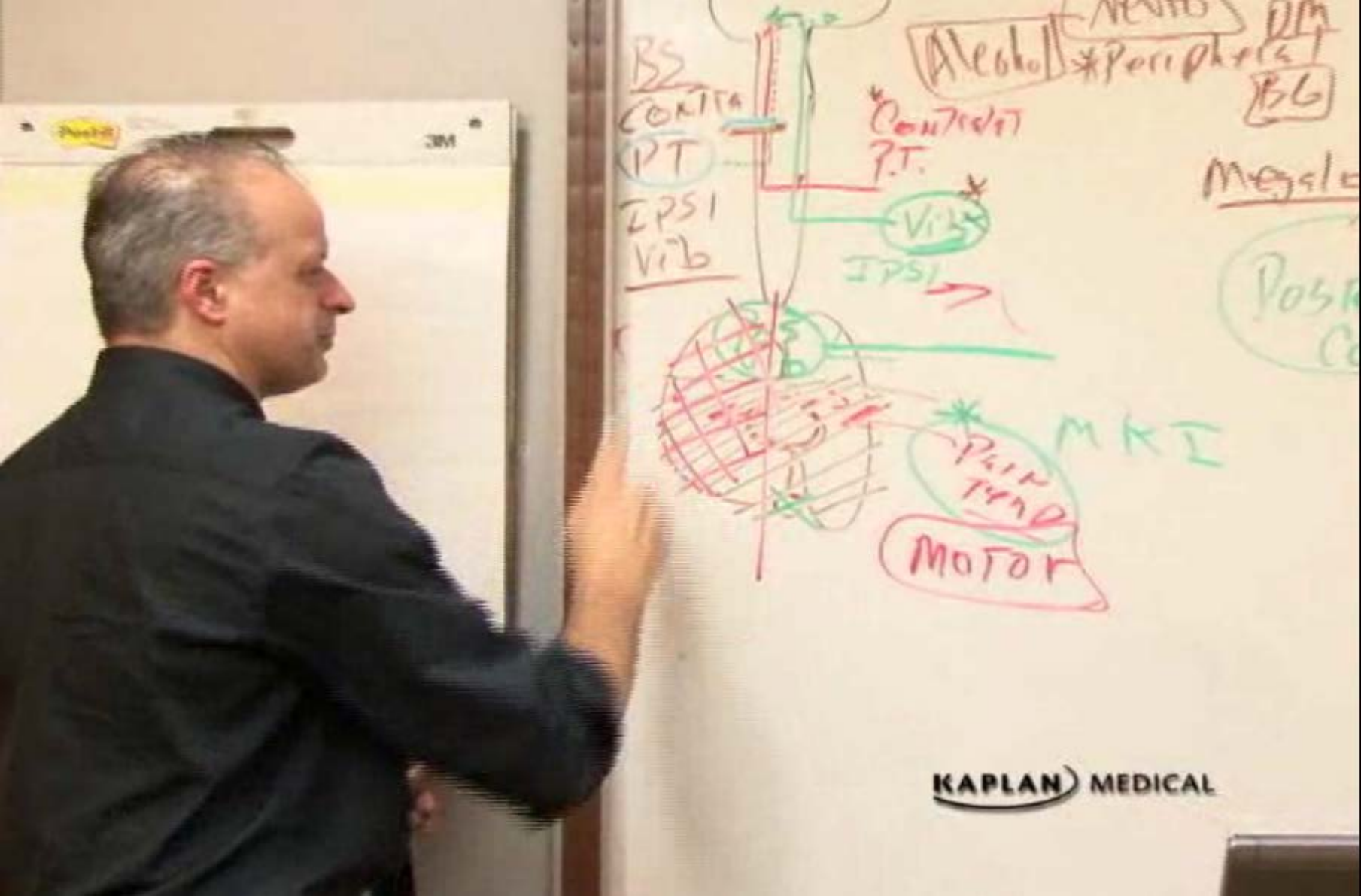
Brown-Sequard Syndrome


BS
CONTRA
IPT
IPSI
Vib
Contrast
P.T.
IPSI
MRI
MOTOR
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Brown-Sequard Syndrome

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Vertigo and Dizziness

Vertigo vs. Dizziness

Sensation	Diagnosis	Etiology
The environment is "spinning" Sensation of movement without actually moving	Vertigo	Neurological
Lightheadedness Feeling like they are going to "black out"	Presyncope	Cardiac

Central vs. Peripheral Disease

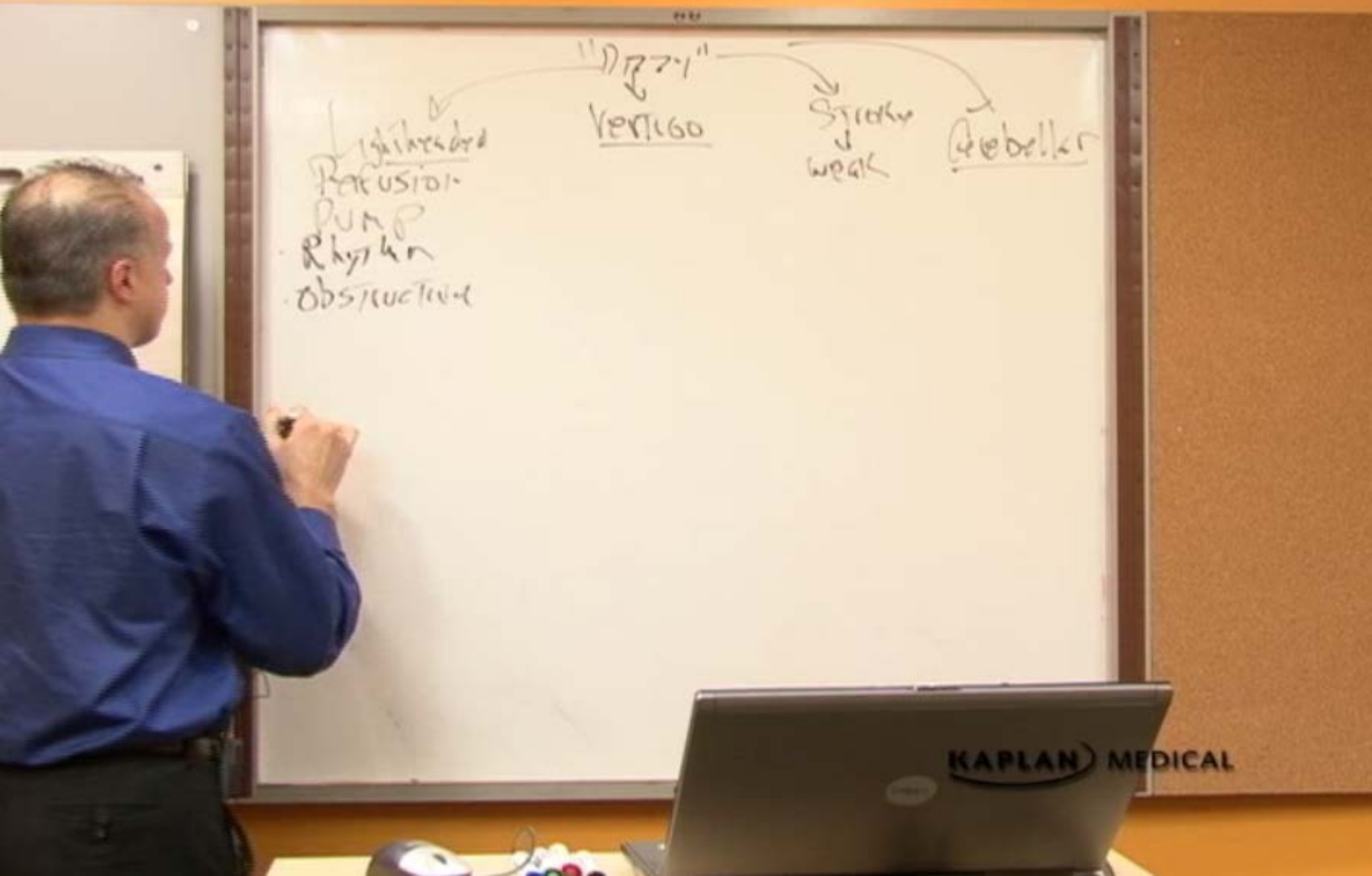
	Central Vertigo	Peripheral Vertigo
Onset	Gradual	Usually sudden
Tinnitus, Hearing Loss	Absent	Present
Neighborhood Signs (diplopia, cortical blindness, dysarthria, extremity weakness/numbness)	Present	Absent
Nystagmus	Pure, vertical, does not suppress with fixation, and multidirectional	Mixed, horizontal, suppresses with fixation, and unidirectional

Vertigo — Etiology

Peripheral Vertigo	• Meniere's Disease– tinnitus, hearing loss and episodic vertigo lasting 1-8 hours • Labyrinthitis– sudden and severe, lasts days with hearing loss and tinnitus • Benign paroxysmal positional vertigo– exacerbated by movement lasting seconds • Perilymphatic fistula– due to trauma

Vertigo — Treatment

- Based on etiology
- Peripheral vertigo and labrynthitis
  - Symptomatic treatment with meclizine or diazepam when severe
- Meniere's Disease
  - Low-salt diet and diuretics, surgery if failure to medical therapy occurs
- Benign paroxysmal positional vertigo
  - Positional maneuvers to dislodge the otolith


Vertigo
Stroke
weak
Cerebellar
Rhythm
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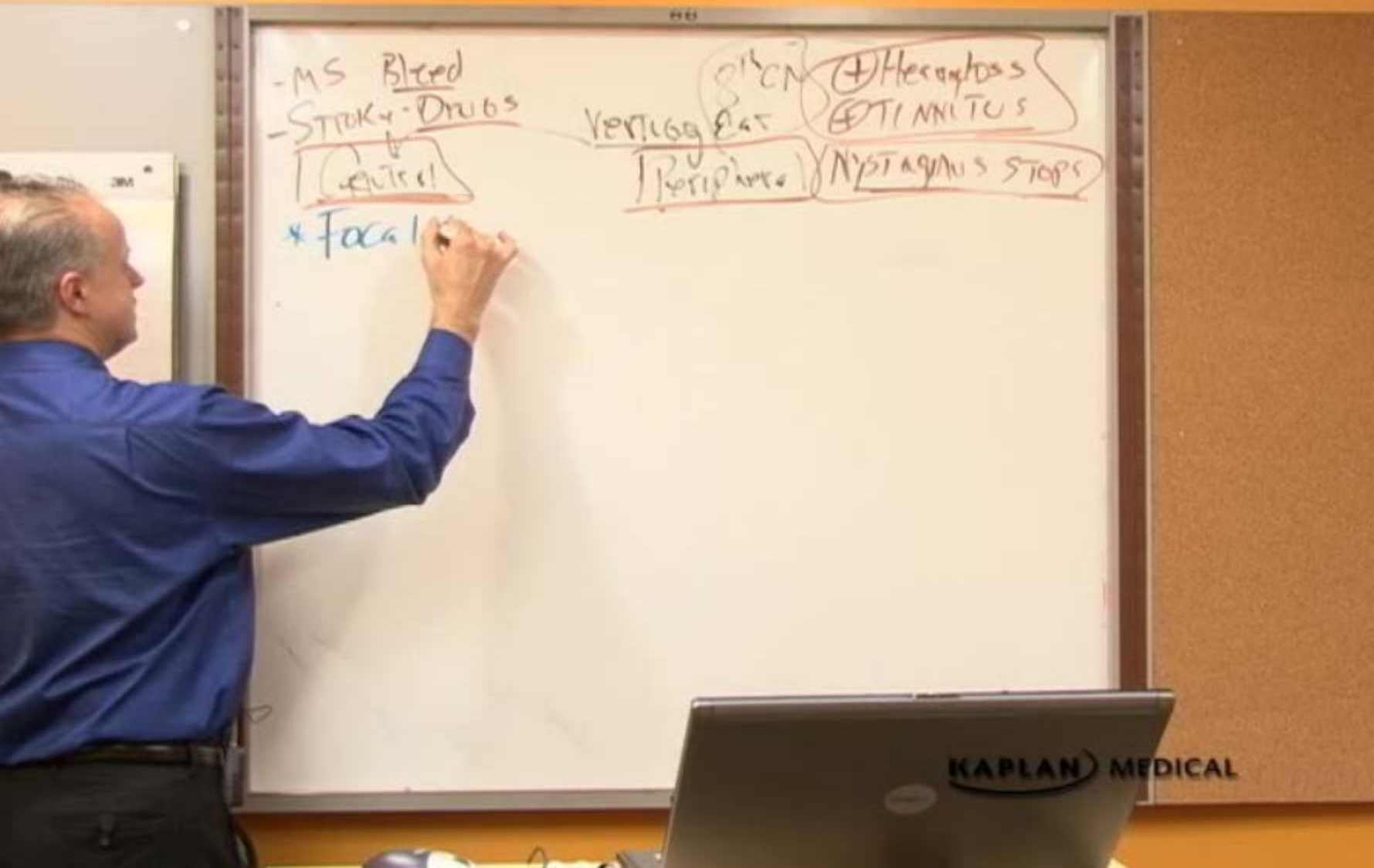
-MS Bleed
-Sticky-Drugs
Central
* Focal
Vertigo
Peripheral
8th CN
+Horizontal
+TINNITUS
Nystagmus stops
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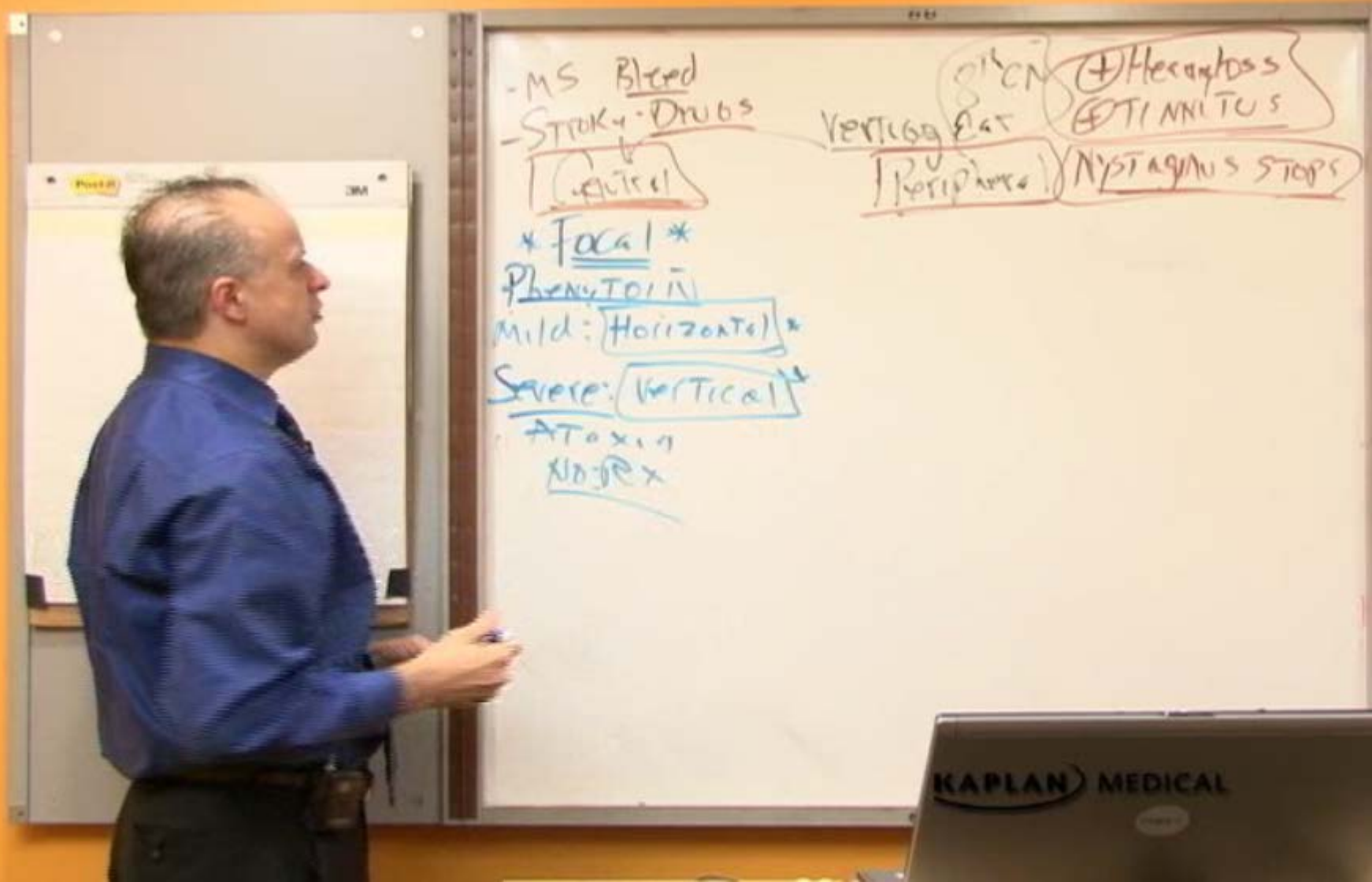
- MS Bleed
- Stroke - Drugs
Central
* Focal *
Phenytoin
Mild: Horizontal
Severe: Vertical
Ataxia
Nystagmus
8th CN
Vertigo Ear
Peripheral
(+) Hearing loss
(+) TINNITUS
Nystagmus stops
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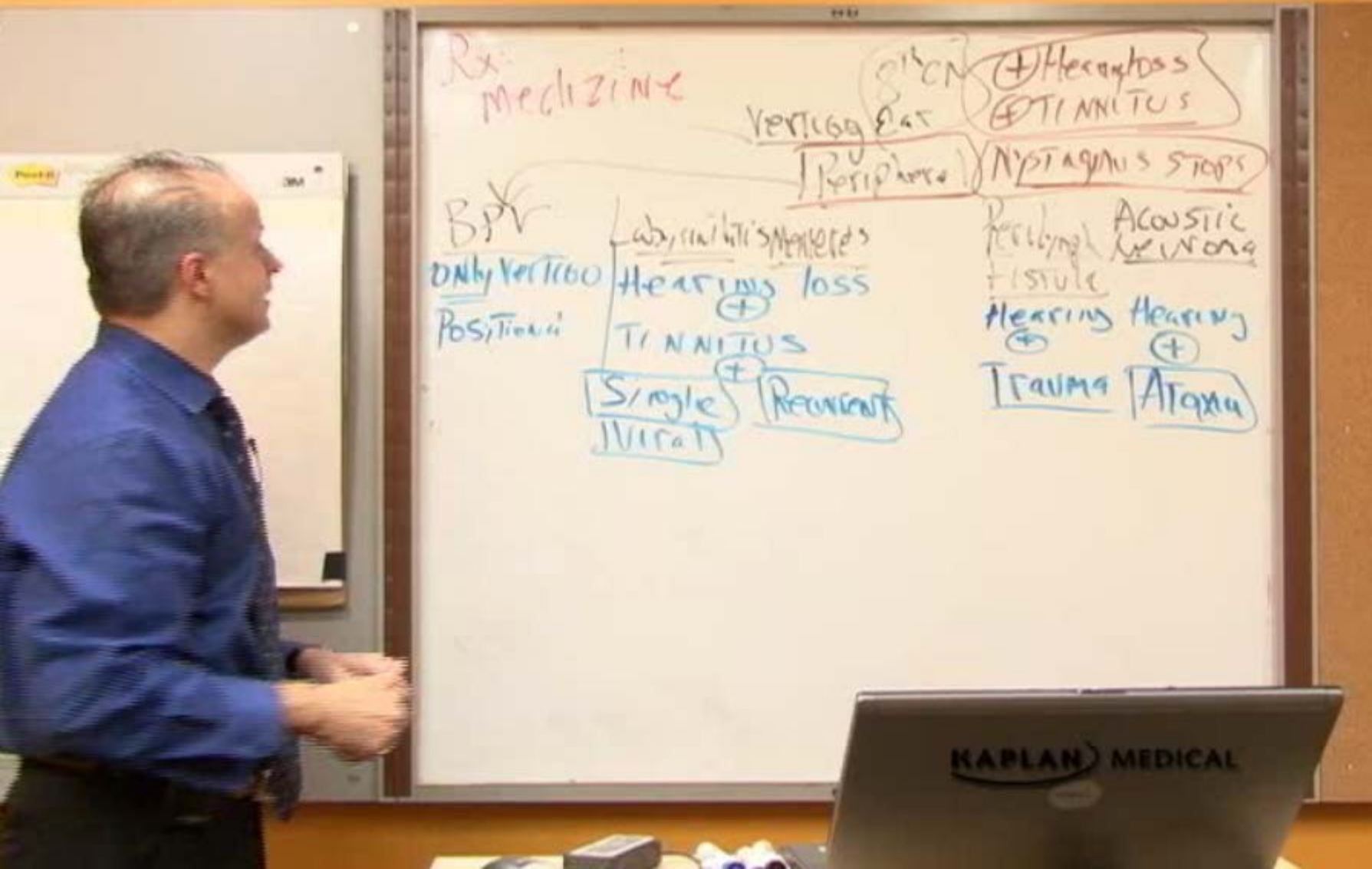
Rx:
Medizine
8th CN
+Hearing loss
+TINNITUS
Vertigo Eat
Peripheral
Nystagmus stops
BPV
Only Vertigo
Positional
Labyrinthitis/Meniere's
Hearing loss
+
TINNITUS
+
Single
Viral
Recurrent
Perilymph
Fistula
Acoustic
Neuroma
Hearing
+
Hearing
+
Trauma
Ataxia
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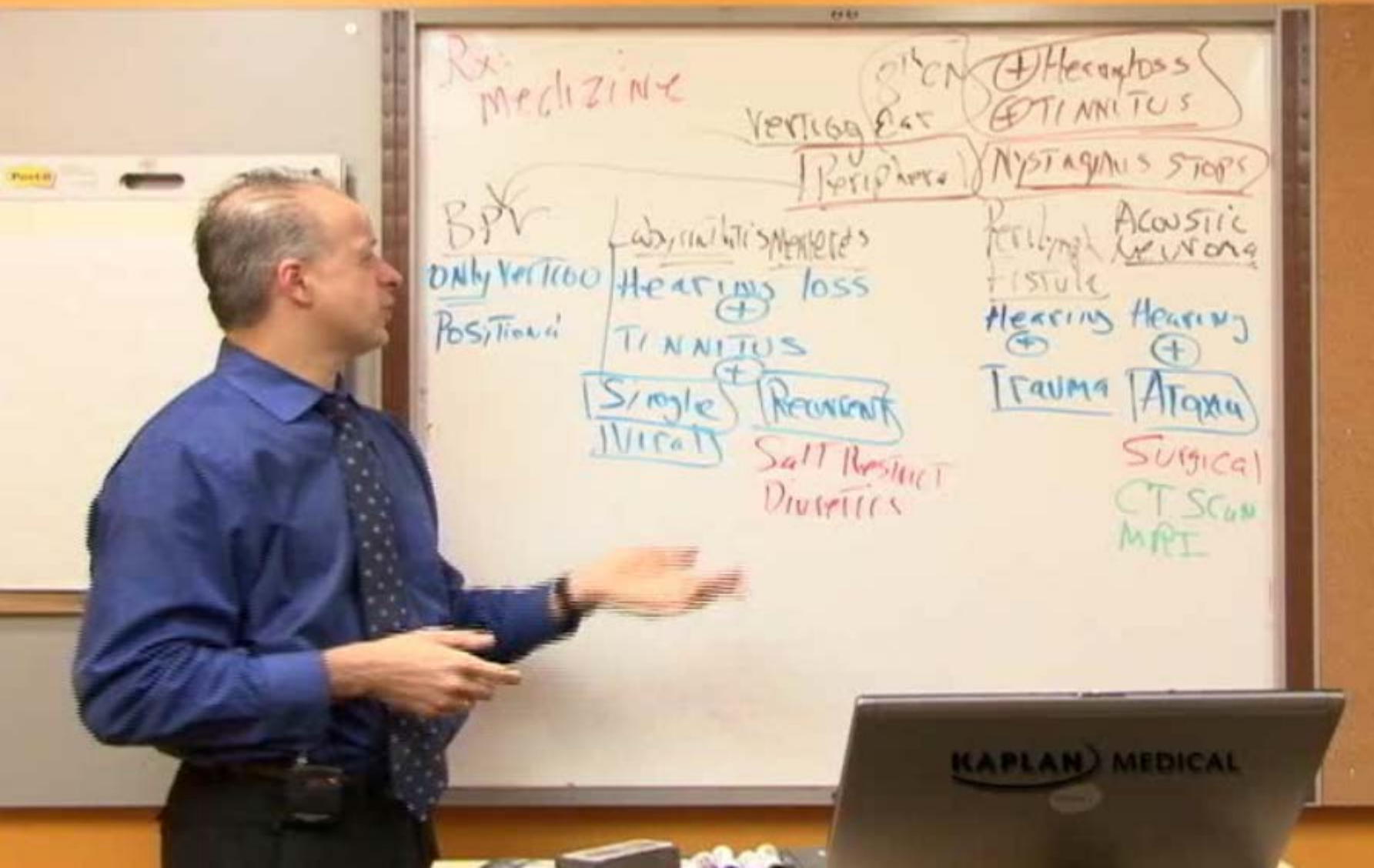
Rx:
Medizine
Vertigo Ear
(+) Hearing loss
(+) TINNITUS
Nystagmus stops
BPV
Only Vertigo
Positional
Hearing loss
(+)
TINNITUS
(+)
Single
Recurrent
Salt Restrict
Diuretics
Trauma
Hearing
(+)
Ataxia
Surgical
CT Scan
MRI
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Vertigo and Dizziness

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Headache

Headache — Etiology

Primary Headaches	***Secondary Headaches***
Migraines	Intracranial hemorrhage
Cluster headache	Brain tumor
Tension headache	Meningitis
	Temporal Arteritis
	Glaucoma

Headache — Differential Diagnosis

- Primary Headaches
  - Usually recurrent
- Secondary Headaches
  - Usually sudden or severe and accompanied by worrisome signs and symptoms

Headache — Differential Diagnosis

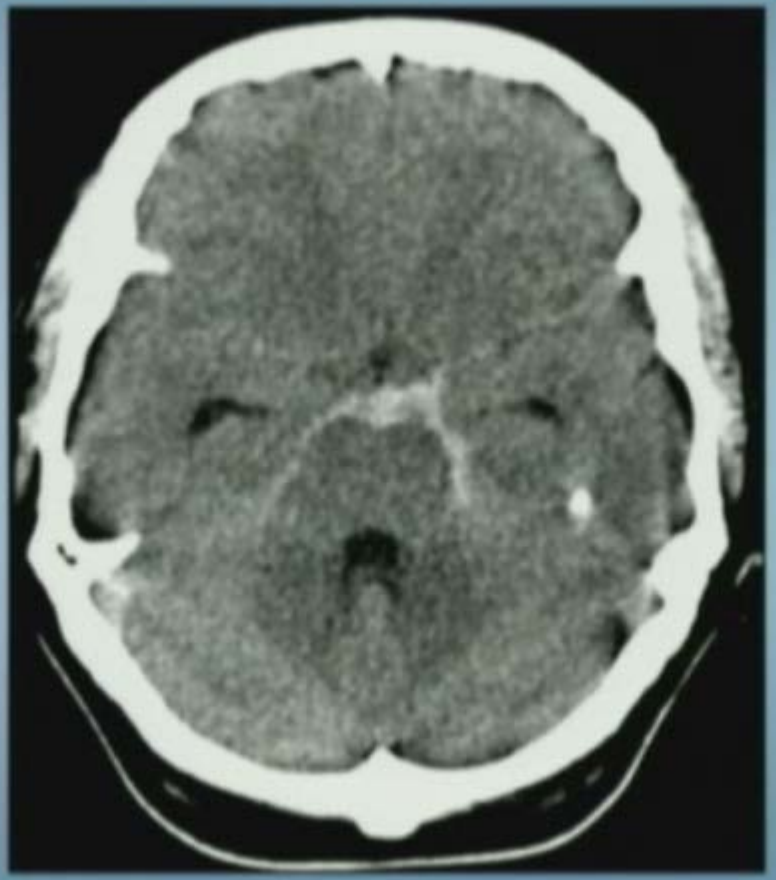
Meningitis	Fever and nuchal rigidity
Intracranial hemorrhage	"Worst headache of my life"
Brain tumor	Deep, dull, aching, wakes them from sleep
Posterior fossa tumors	Vomiting precedes headache by weeks. Worse with bending, lifting or coughing
Temporal arteritis	Unilateral pounding headache with lancinating pain and visual changes

Headache — Differential Diagnosis

- Once serious pathology (secondary causes) are ruled out, proceed to investigate primary causes

Migraine headache	Pulsatile, throbbing, unilateral, aggravated by movement, light or sound
Tension headache	Tight, band like, bilateral with tightness of the posterior neck muscles; builds slowly and may last days
Cluster headache	Excruciating, unilateral, periorbital lasting up to 90 mins. Associated with rhinorrhea, red eye, lacrimation, nasal congestion and nausea

“The worst headache of my life”



Migraine Headache — Treatment

Abortive Treatments	***Prophylactic Treatments***
NSAIDs, aspirin, acetaminophen	Beta-blockers
Triptans	Calcium channel blockers
Ergotamine derivatives	Tricyclics
	SSRIs
	Valproic acid
	Topiramate

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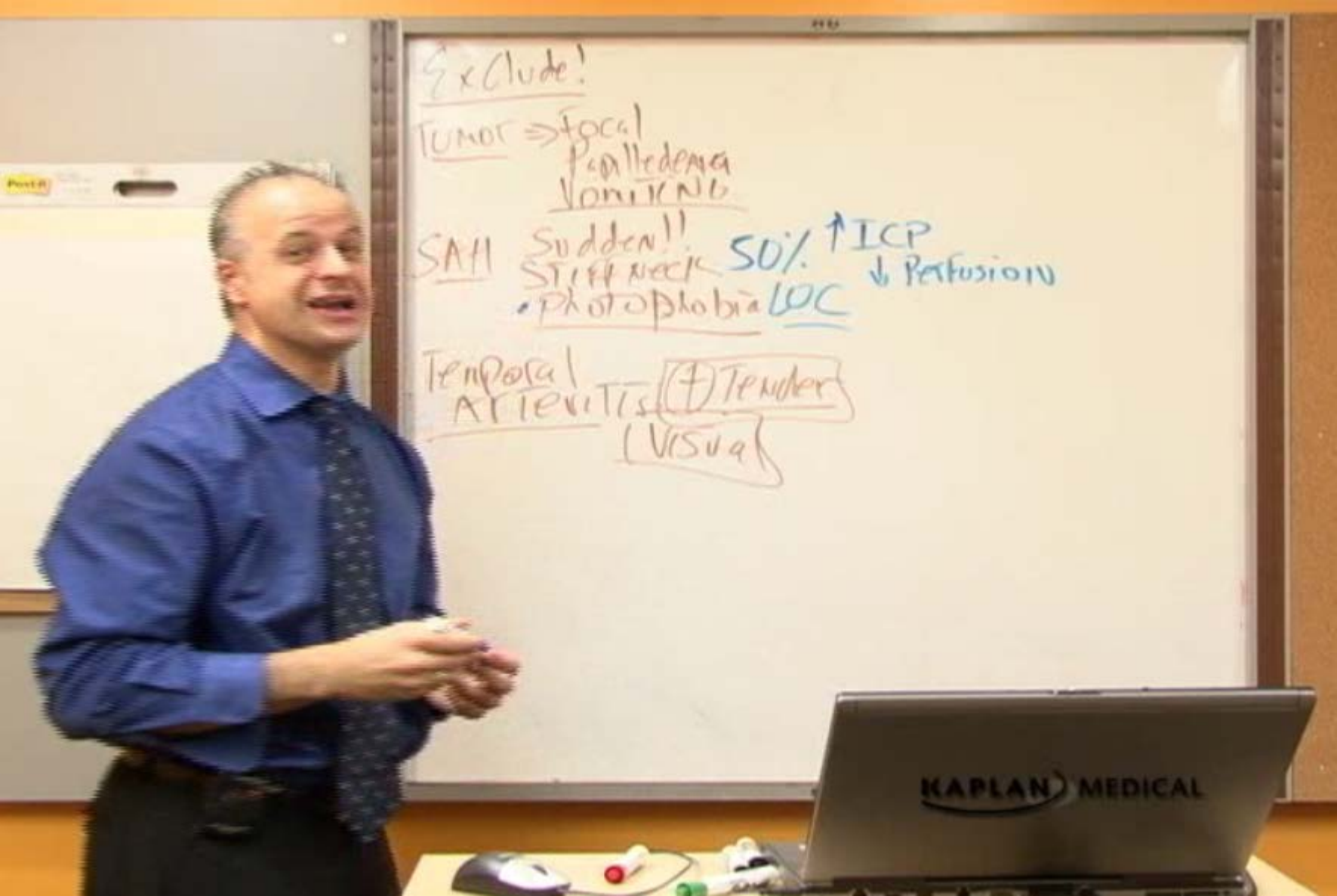
Exclude!
TUMOR ⇒ focal
papilledema
vomiting
SAH Sudden!!
stiff neck
• photophobia
50%
↑ICP
↓ Perfusion
LOC
Temporal
Arteritis
(+) Tender
(Visual
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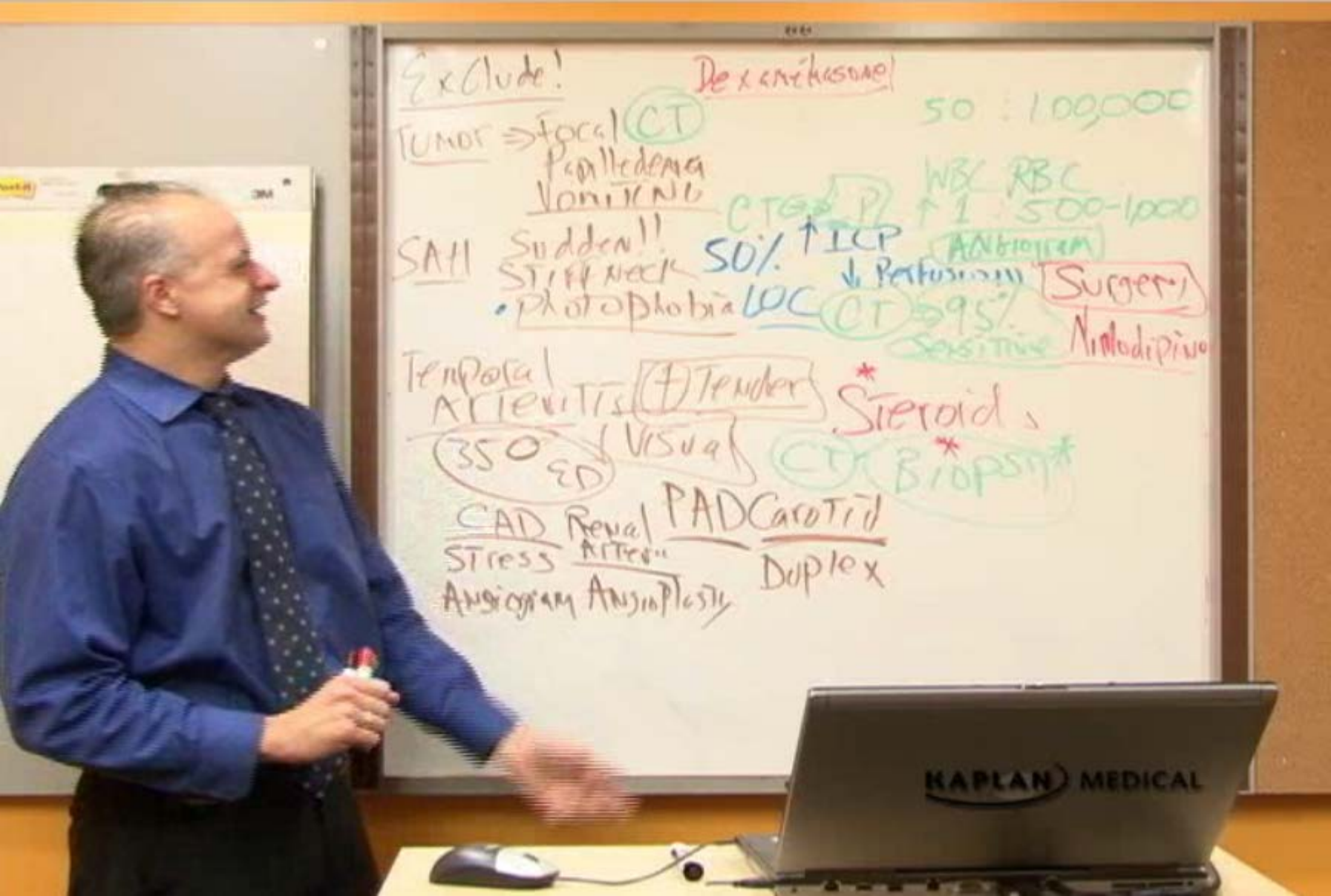
Exclude!
Dexamethasone
50 : 100,000
TUMOR ⇒ focal
CT
Papilledema
Vomiting
WBC RBC
↑ 1 : 500-1000
SAH
Sudden!!
Stiff neck
Photophobia
50%
↑ ICP
LOC
CT ⇒ 95%
Sensitive
Surgery
Nimodipine
Temporal
Arteritis
(+) Tender
Steroid
350 ED
Visual
CT
Biopsy
CAD
Stress
Renal
Artery
Angiogram
Angioplasty
PAD
Carotid
Duplex
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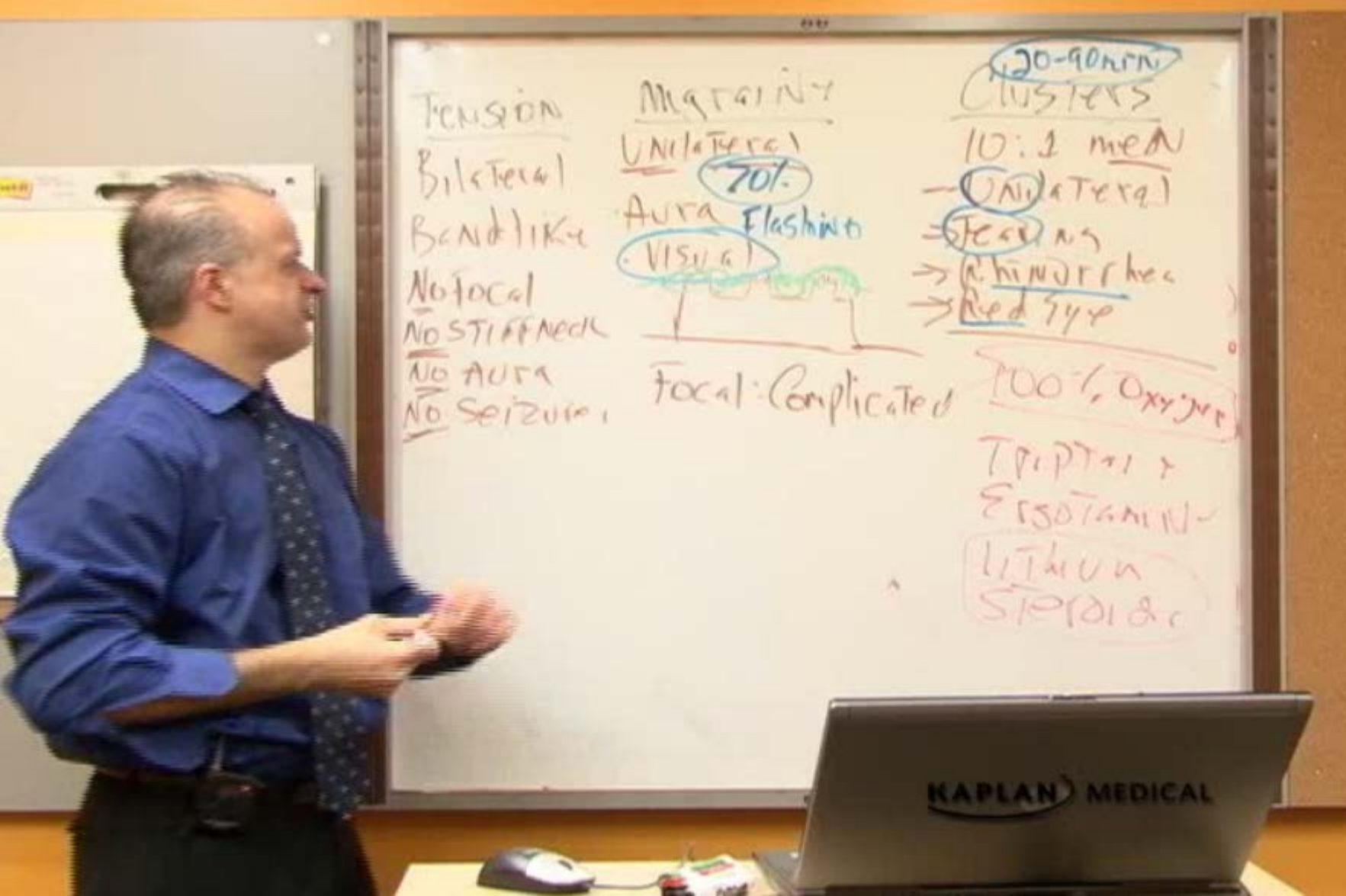
TENSION
Bilateral
Bandlike
No focal
NO STIFF NECK
NO AURA
NO Seizure
MIGRAINE
UNILATERAL
70%
AURA Flashing
Visual
Focal: Complicated
CLUSTERS
10:1 MEN
UNILATERAL
Tearing
Rhinorrhea
Red Eye
100% Oxygen
Triptan +
Ergotamine
Lithium
Steroids
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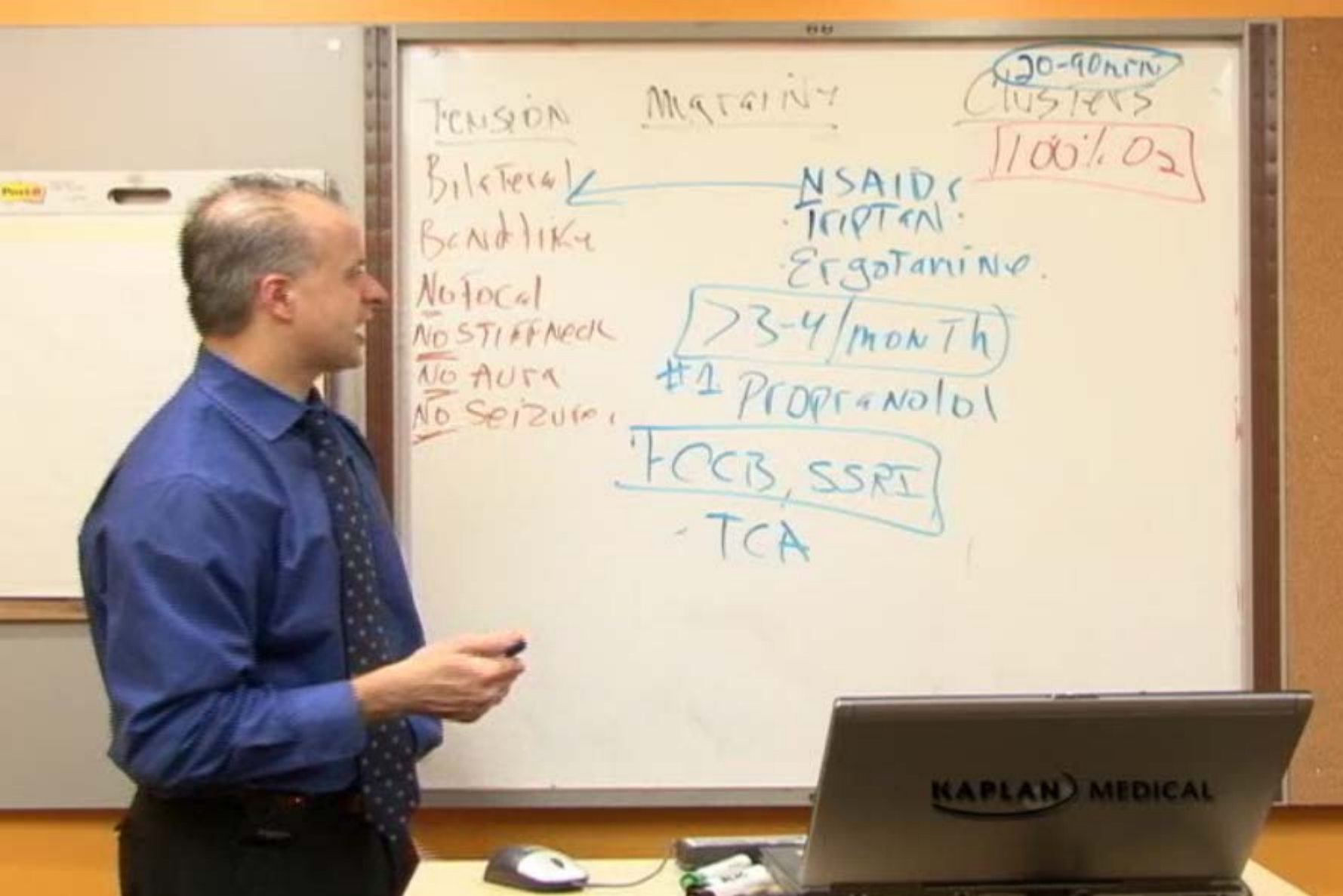
TENSION
MIGRAINE
CLUSTERS
15-90 MIN
100% O2
Bilateral
Band like
NSAIDs
TRIPTANS
Ergotamine
No focal
NO STIFF NECK
NO AURA
NO Seizure
>3-4/MONTH
#1 PROPRANOLOL
FCCB, SSRI
TCA
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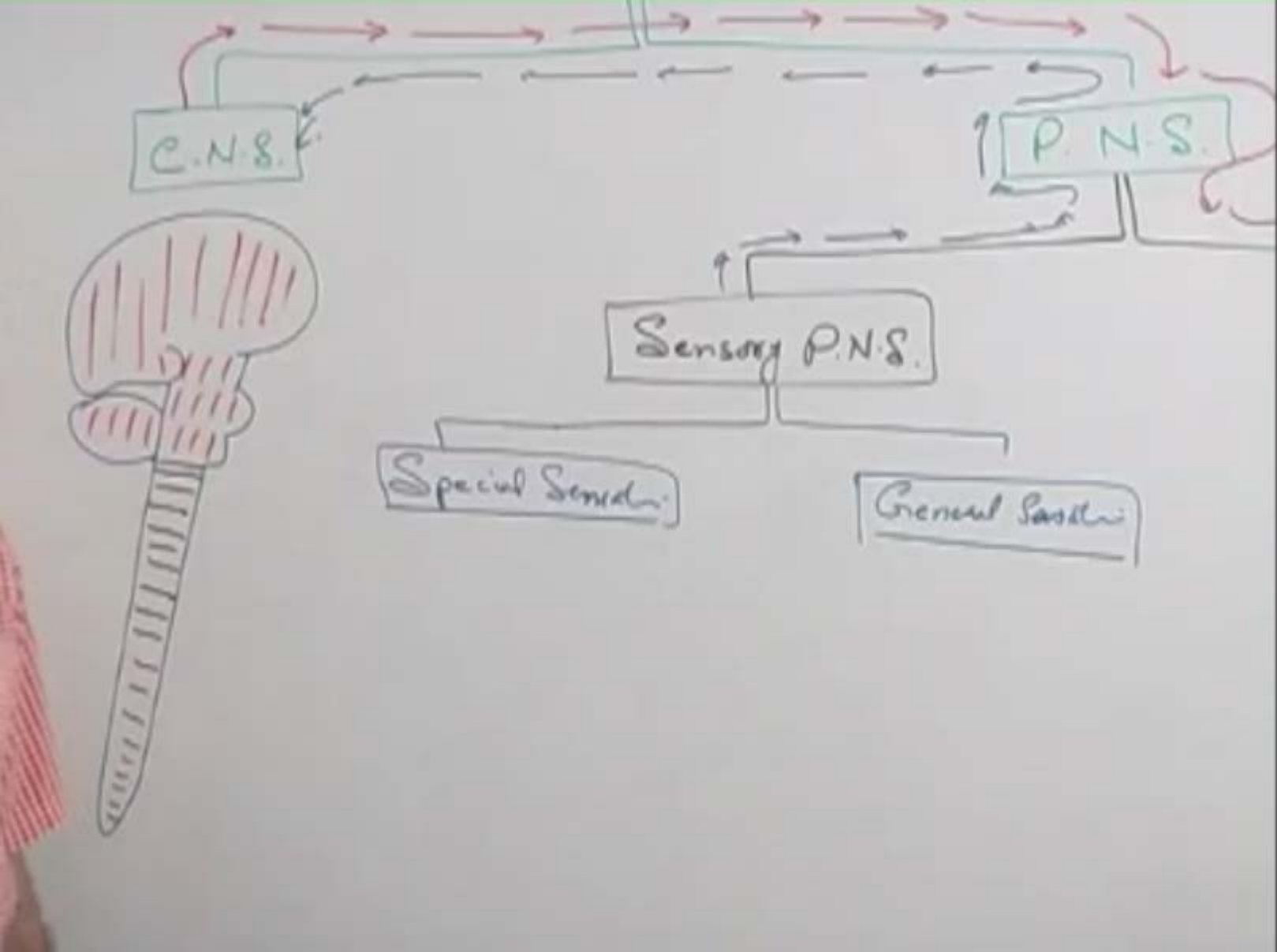
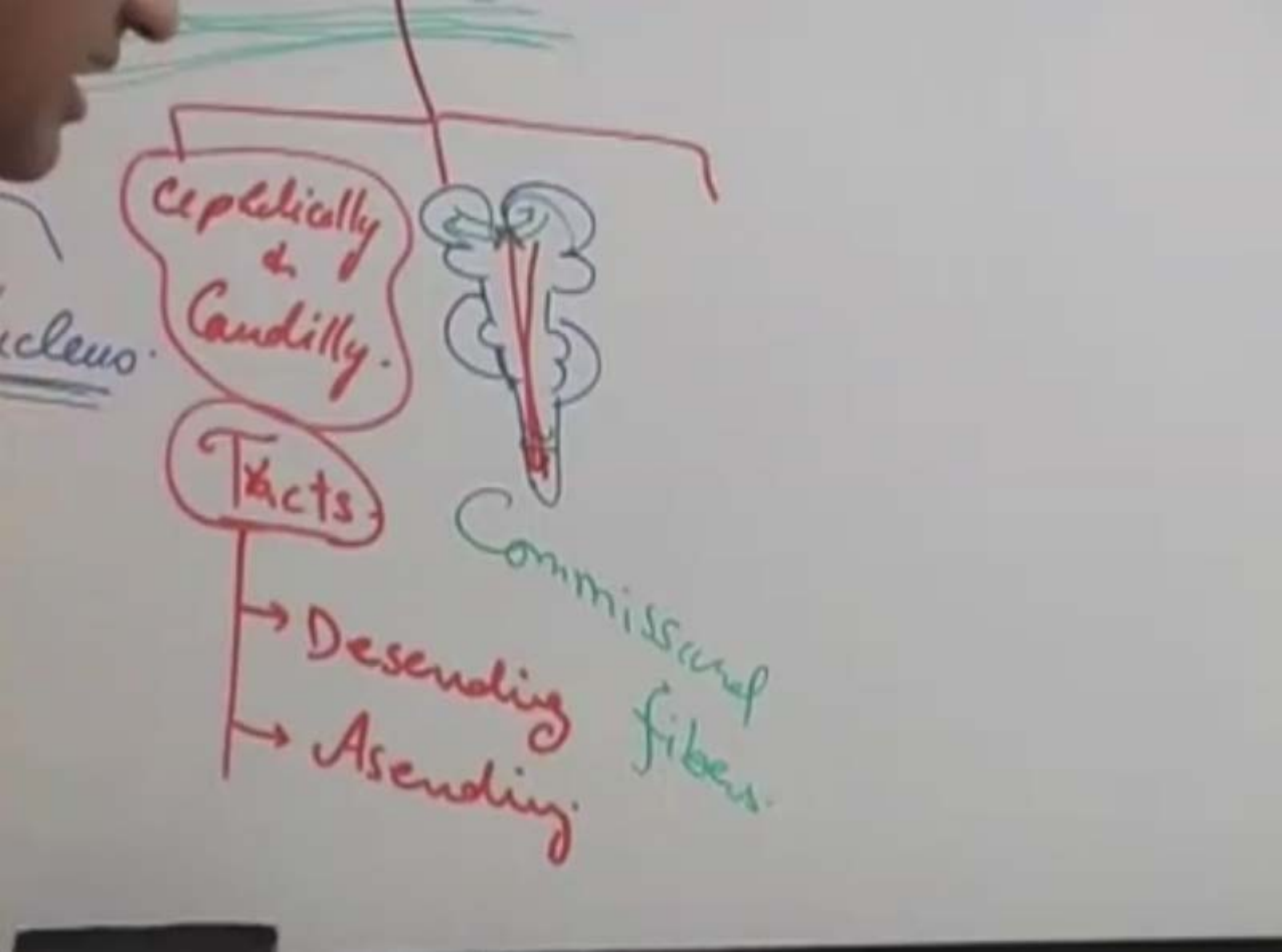
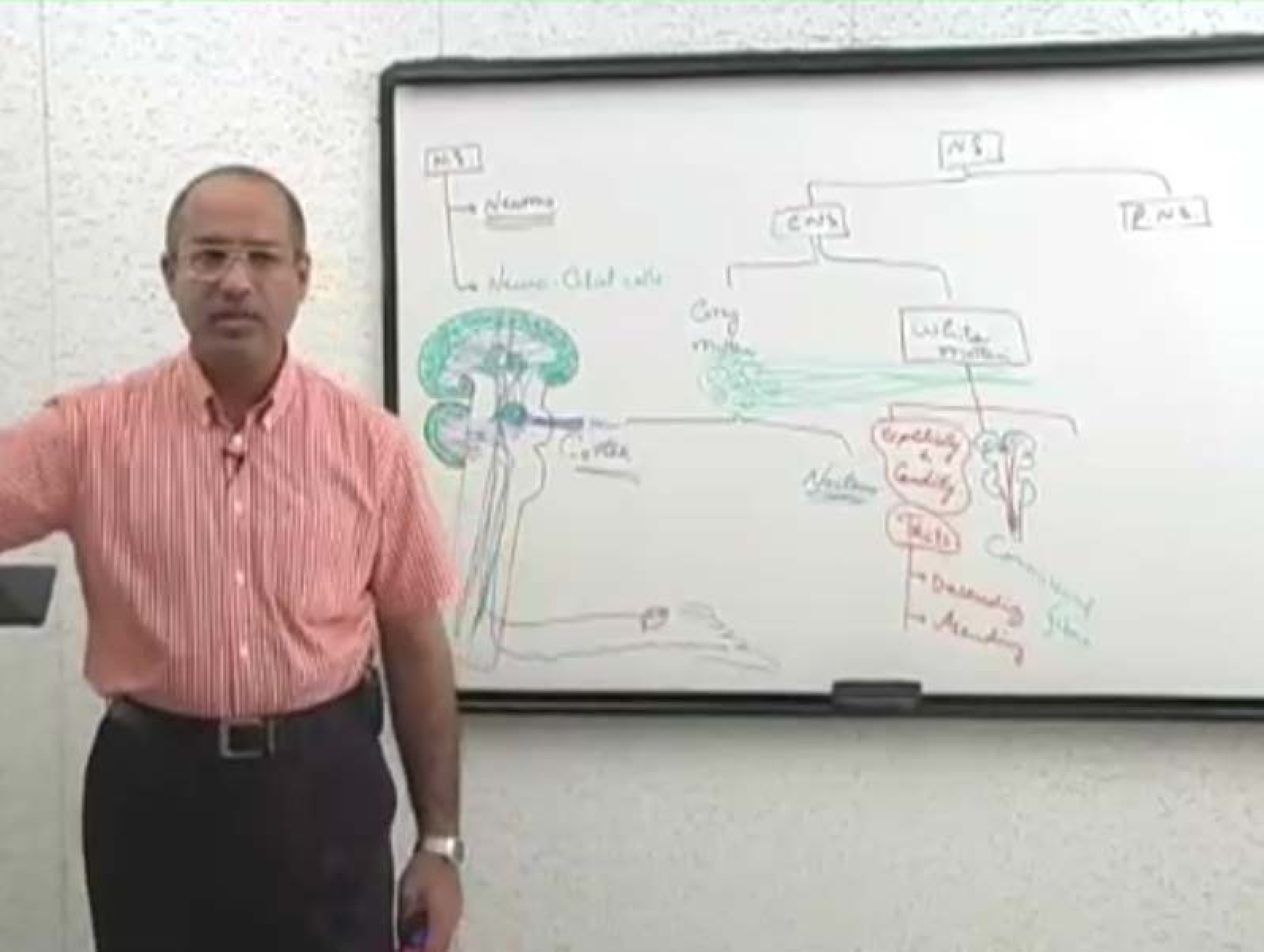
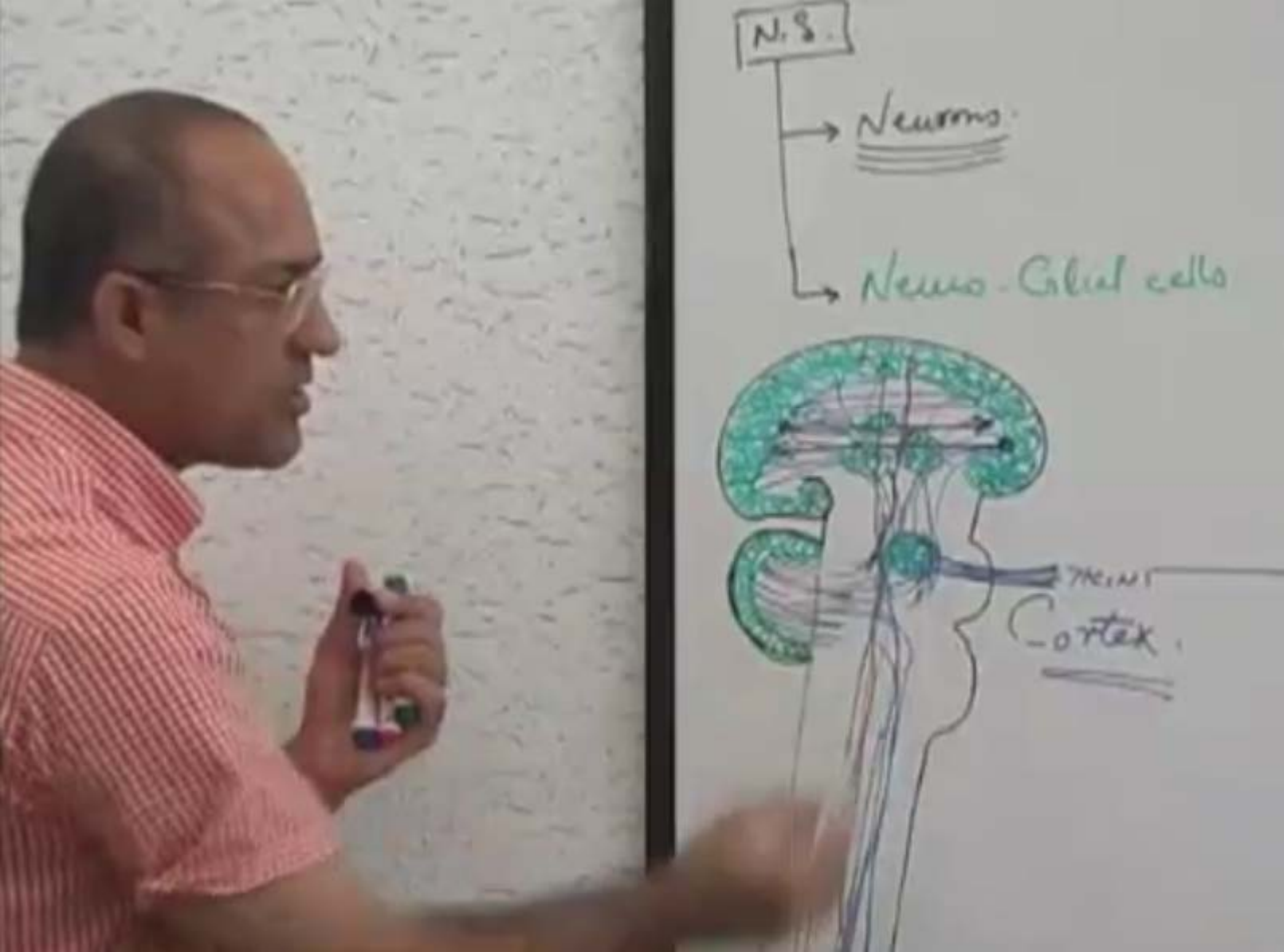
Headache

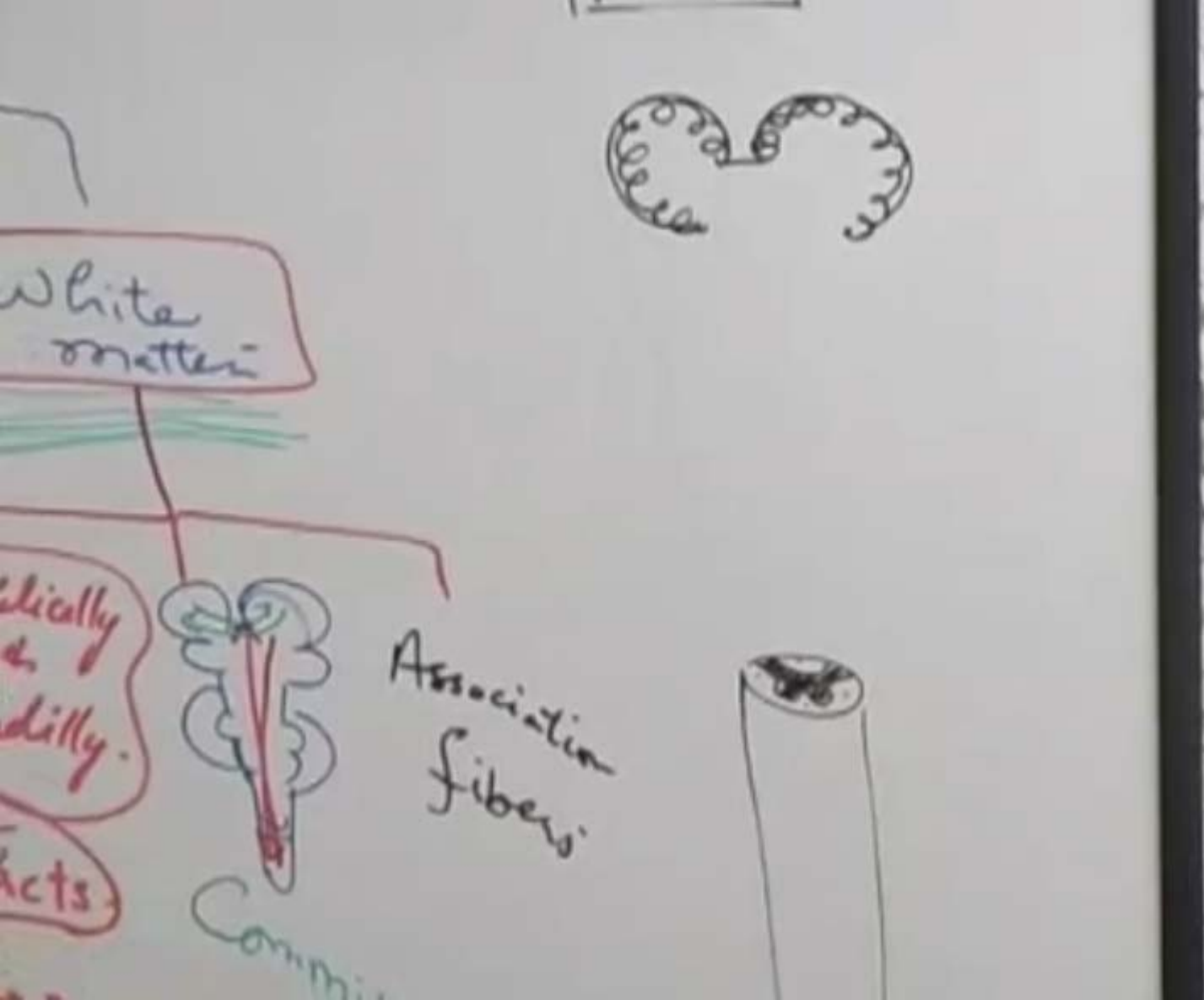
END

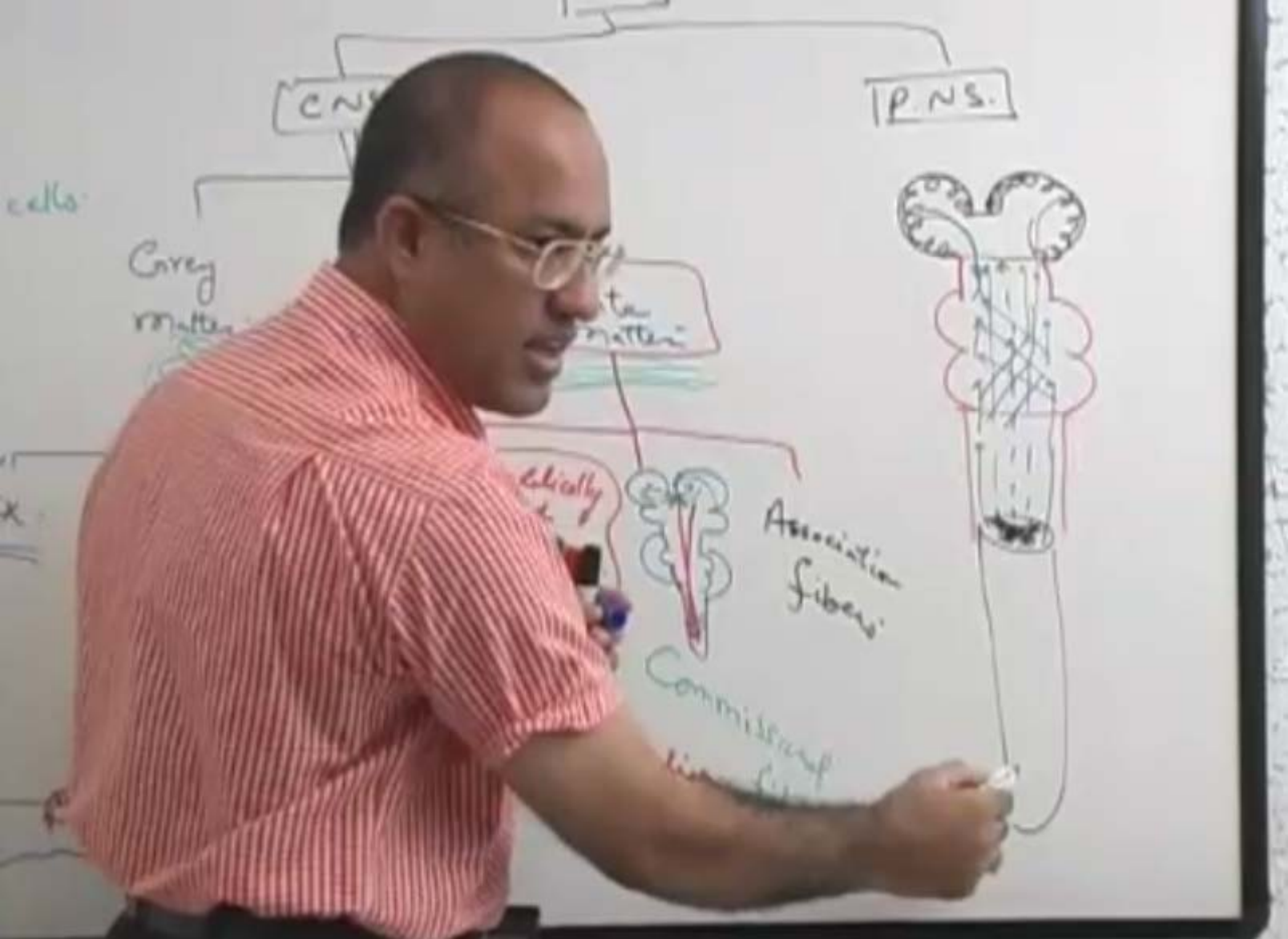

C.N.S.
P.N.S.
Sensory P.N.S.
Special Sensation
General Sensation

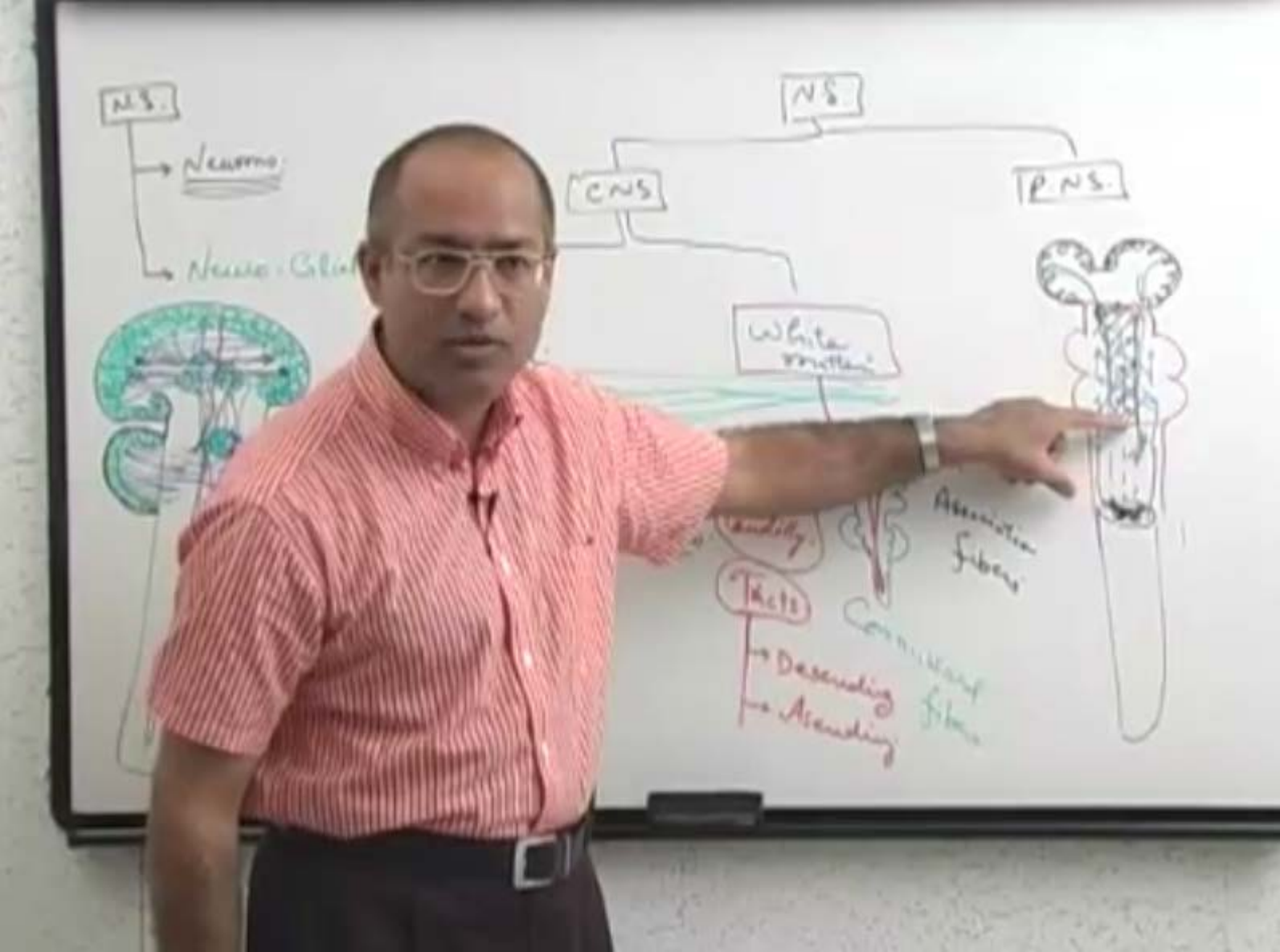

cephalically & caudally.
Tracts
Descending
Ascending
Commissural fibers.

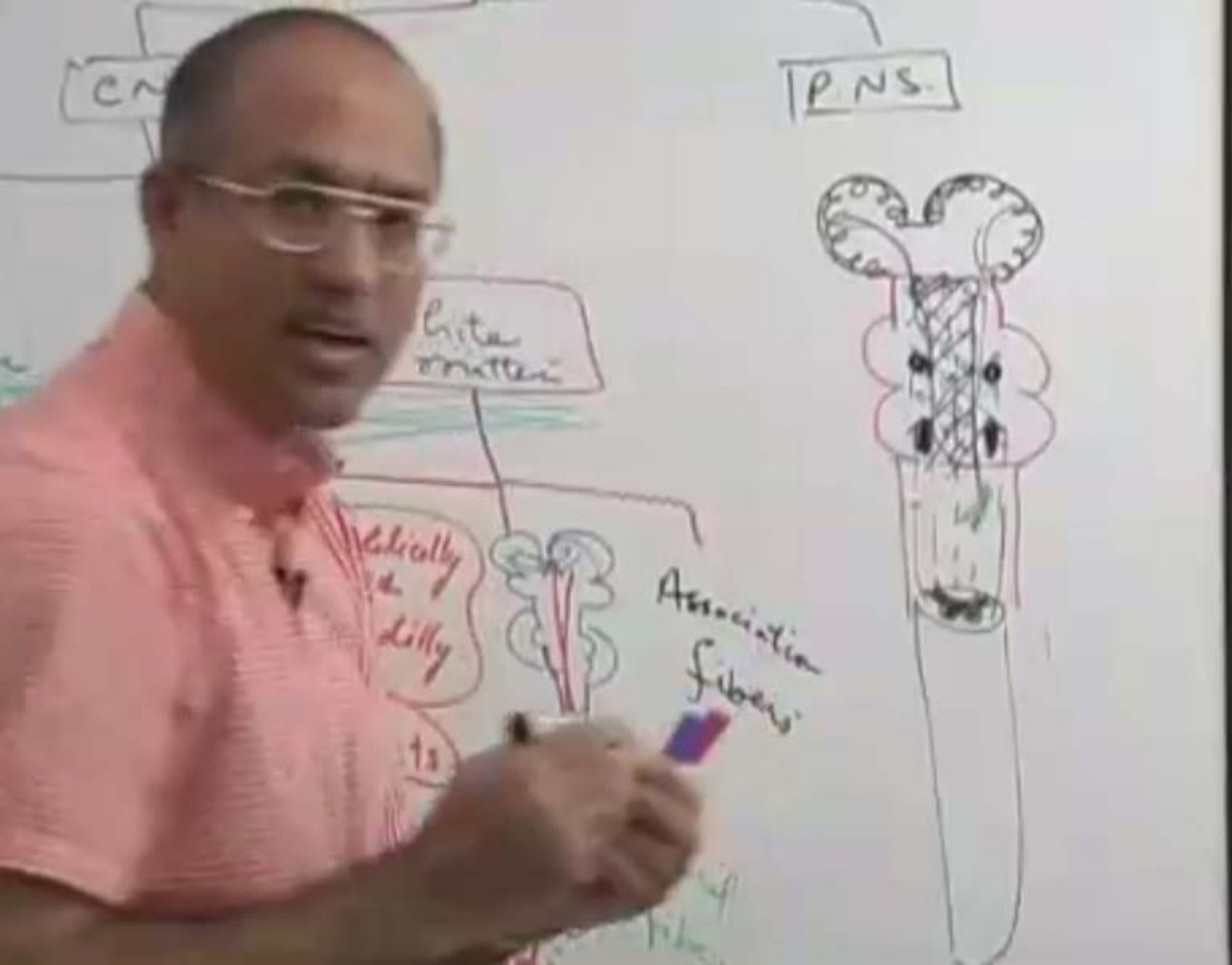

N.S.
Neurons
N.S.
CNS
P.N.S.
White Matter
Cortex
Nucleus
Tracts
Descending
Ascending


N.S.
Neurons
Neuro-Glial cells
Cortex


White matter
Association fibers


P.N.S.
Grey
Association
fibers
Commissural


N.S.
Neurons
N.S.
C.N.S.
P.N.S.
White matter
Tracts
Descending
Ascending
Association fibers


P.N.S.
Association fibers

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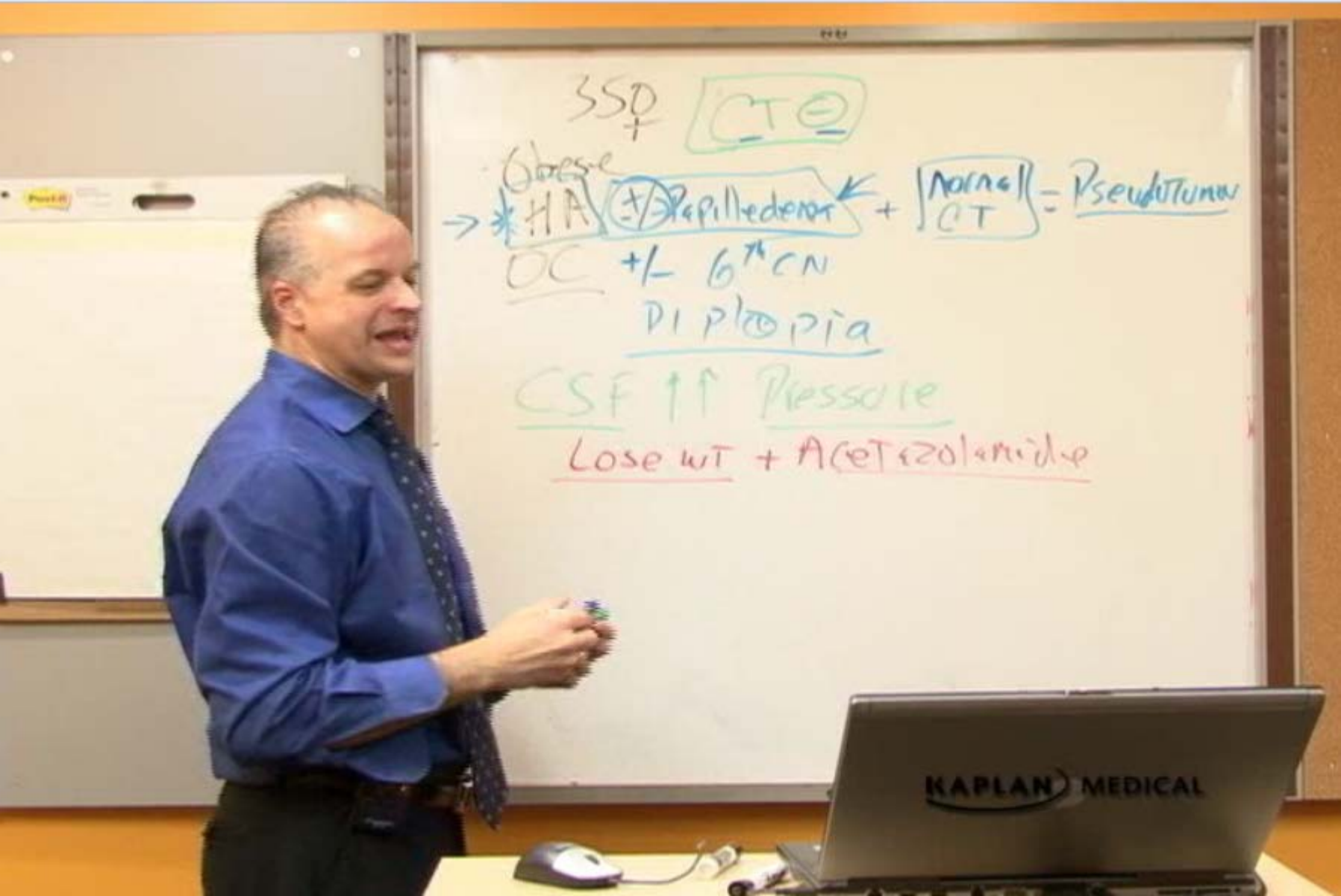
Pseudotumor Cerebri


SSD +
CT −
Obese
HA
+/− Papilledema
+
Normal CT
= Pseudotumor
OC
+/− 6th CN
Diplopia
CSF ↑↑ Pressure
Lose wt + Acetazolamide
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Pseudotumor Cerebri

End

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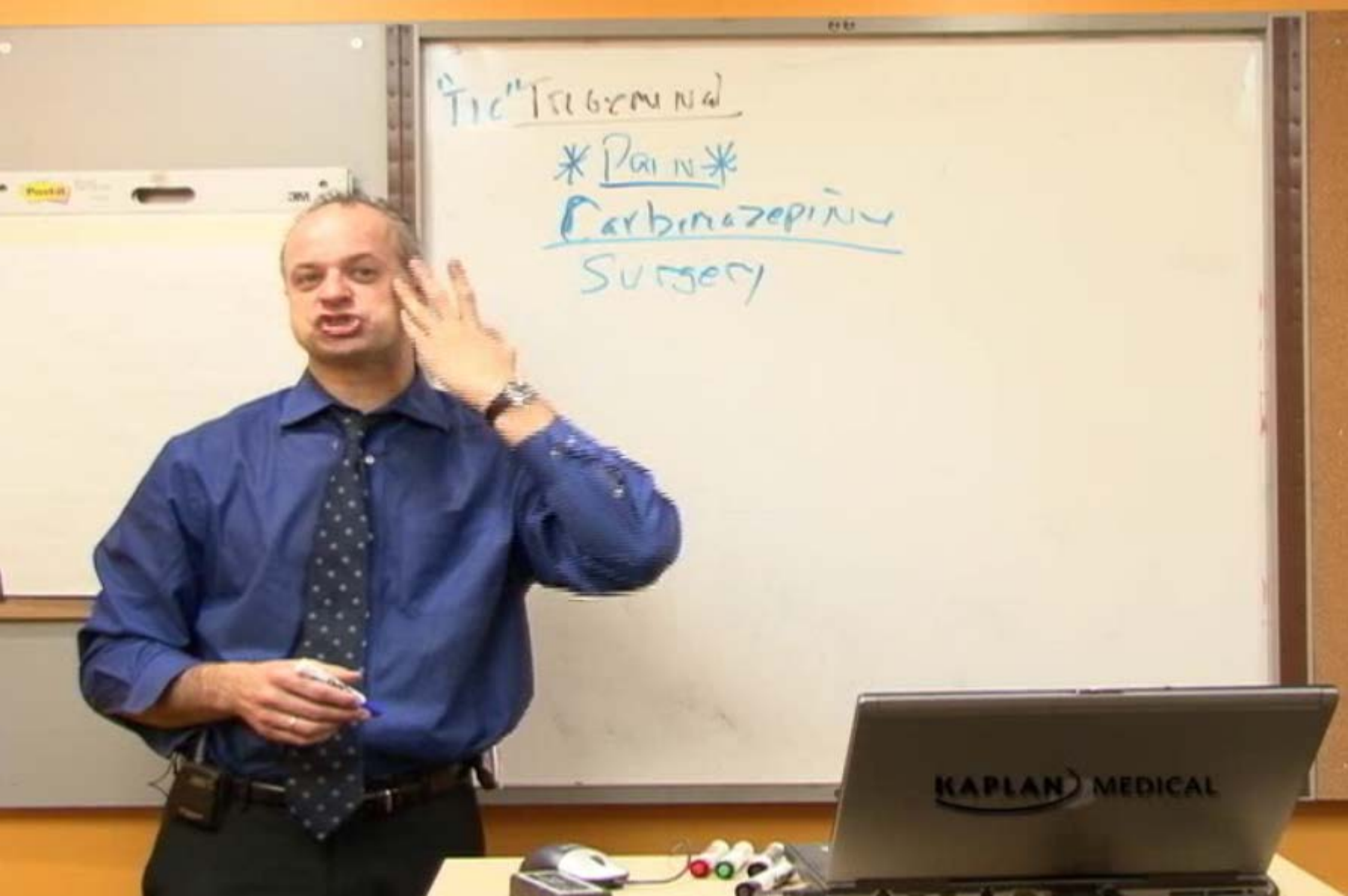
Trigeminal Neuralgia


"TIC" TRIGEMINAL
* PAIN *
Carbamazepine
Surgery
KAPLAN MEDICAL

KAPLAN

MEDICAL

Trigeminal Neuralgia

End

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Ch. 5: Guillian Barrè Syndrome

Guillian Barrè Syndrome — Etiology

- Autoimmune destruction of myelin
- History of infection 1-3 weeks prior to onset
  - Respiratory or GI system
  - *Campylobacter jejuni*, human herpesvirus, cytomegalovirus and Epstein-Barr virus have all been implicated
- Due to molecular mimicry: the immune system attacks self-antigens as foreign-antigens

Guillian Barrè Syndrome — Presentation

- Rapid development of weakness starting in the lower extremities and moving upwards
- Absent reflexes
- Progression over hours to days
- Legs > arms
- Pain and tingling sensations
- Presence of fever, constitutional symptoms or bladder dysfunction should raise questions to the diagnosis

Guillian Barrè Syndrome — Diagnosis

- Best initial test ⟹
  Lumbar puncture
  - Changes occur >48 hours after onset
  - Increased protein without increased cell count
- Most accurate test ⟹
  Electromyelography (EMG)
  - Detects demyelination of peripheral nerves

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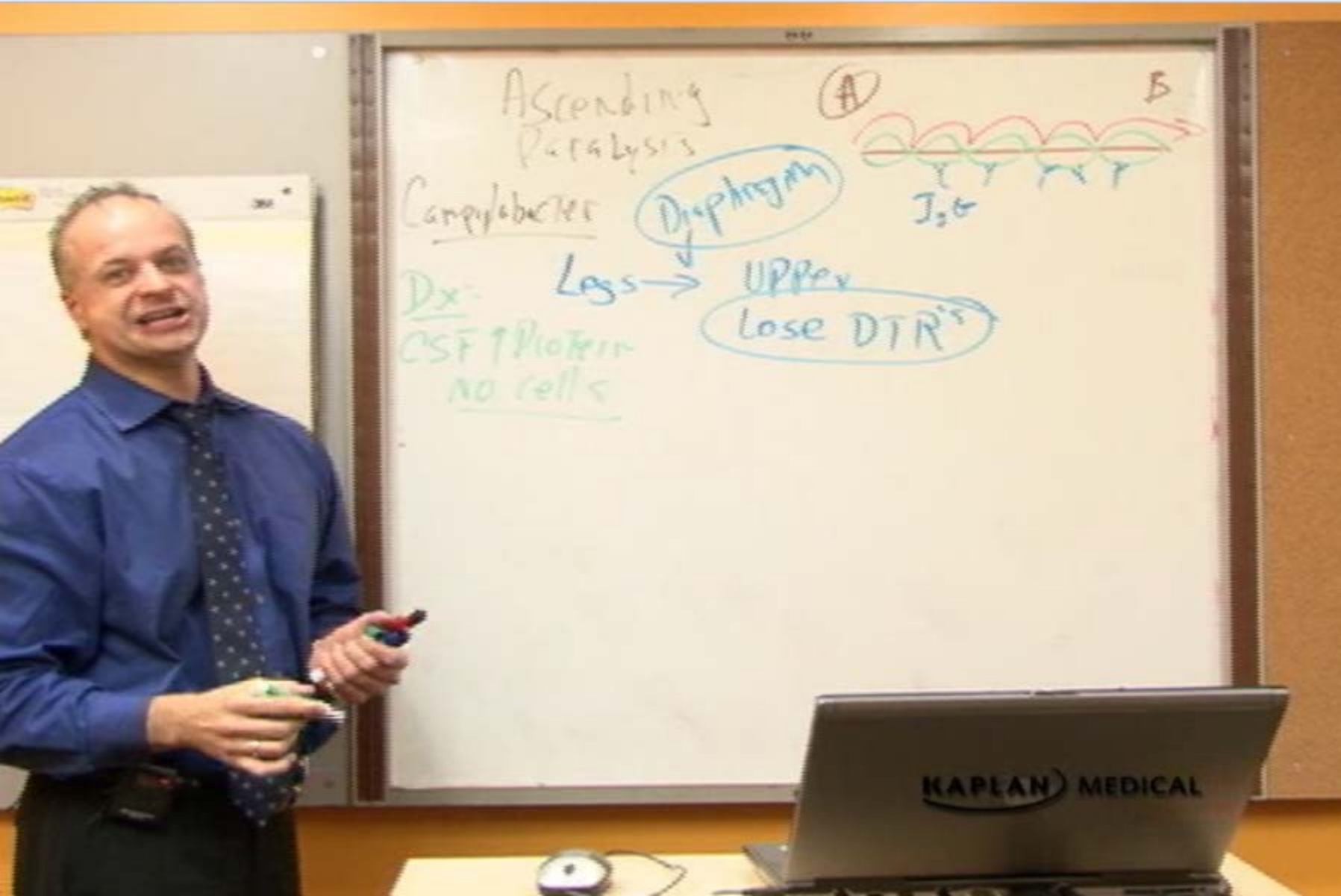
Guillian Barrè Syndrome — Treatment

- Should be started ASAP
  - Becomes ineffective 2 weeks after onset of symptoms
- Intravenous immunoglobulin or plasmapheresis
  - Both are equally effective
- Monitor for impending respiratory failure and intubate with mechanical ventilation if required


Ascending Paralysis
A
B
Campylobacter
Diaphragm
Legs → UPPERS
Lose DTR's
Dx:
CSF ↑ Protein
NO cells
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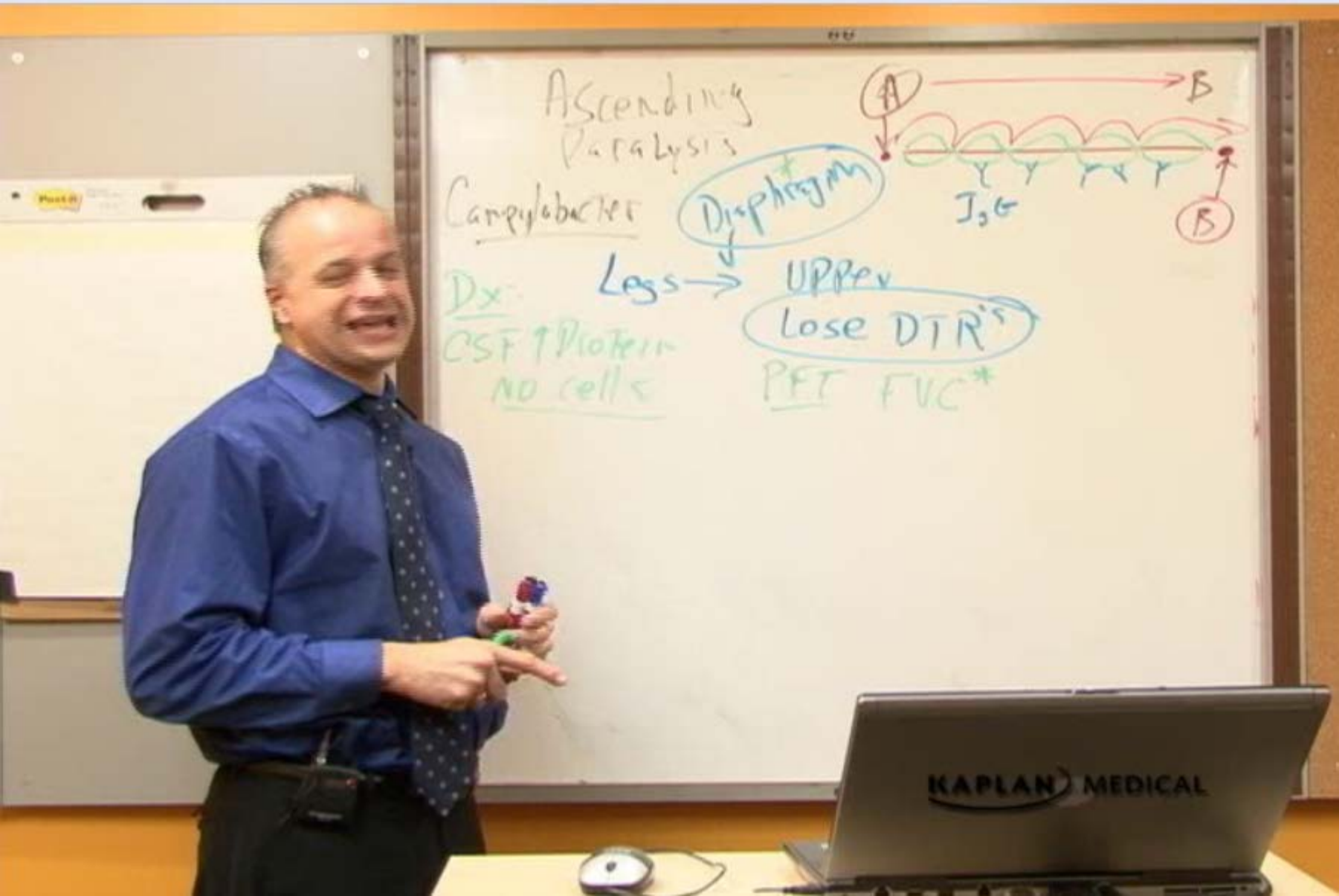
Ascending
Paralysis
Campylobacter
Diaphragm
Legs
Dx:
CSF ↑ Protein
NO cells
UPPer
Lose DTR's
PFT FVC*
A
B
B
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Ascending
Paralysis
A
B
Diaphragm
UPRev
Lose DTR's
Rx
IVIG
Plasmapheresis
Steroids
KAPLAN MEDICAL

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MEDICAL

Ch. 5: Guillian Barrè Syndrome

END

KAPLAN MEDICAL

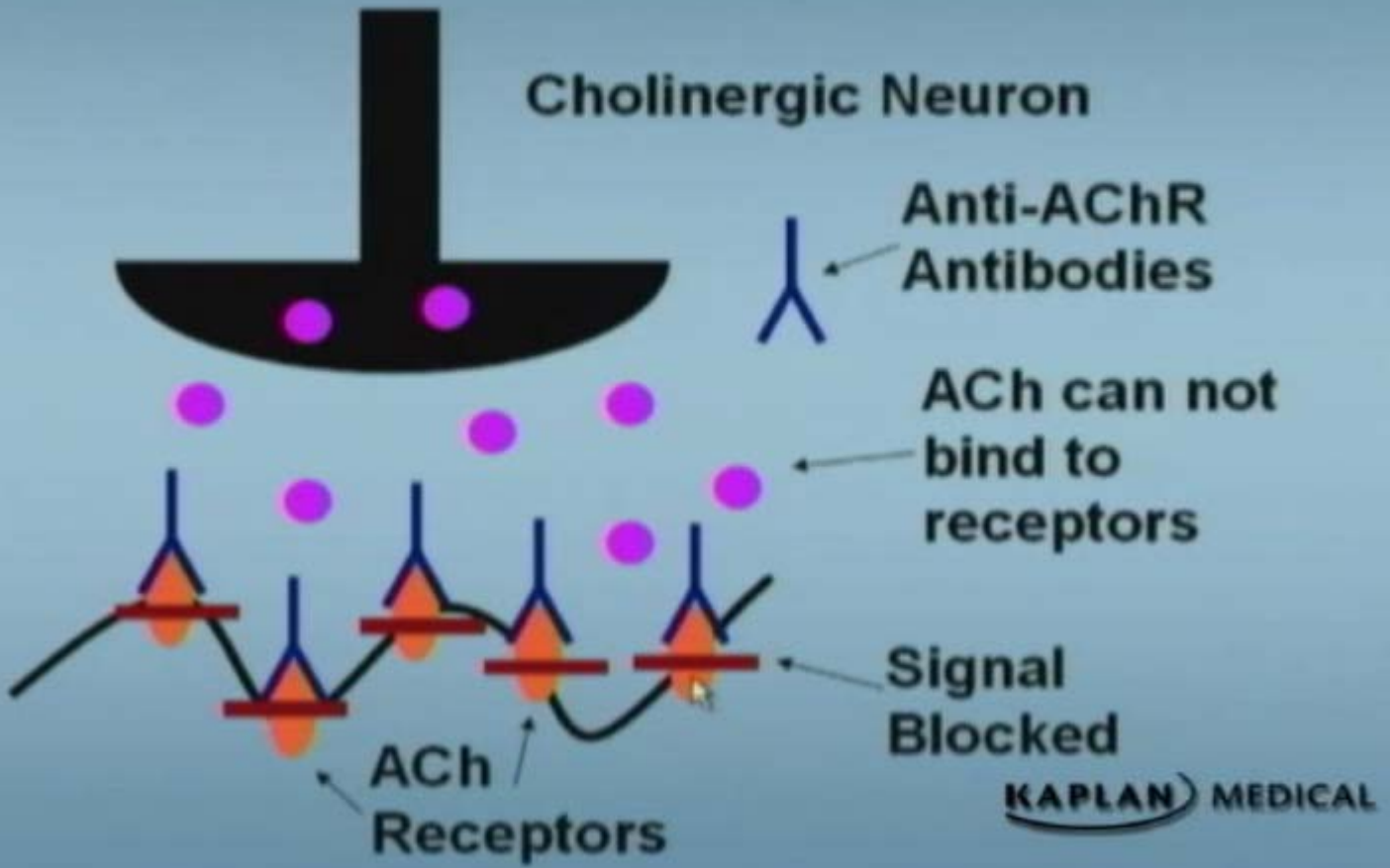
Ch. 6: Myasthenia Gravis

Myasthenia Gravis — Etiology

Acetylcholine auto-antibodies directed at the neuromuscular junction

Decreased number of active and functional acetylcholine receptors at the post-synaptic membrane

Myasthenia Gravis — Etiology



Myasthenia Gravis — Clinical Presentation

- Diplopia, ptosis and difficulty swallowing
- Nasal speech
- “Snarling” smile
- Weakness may become generalized and asymmetric
- Deep tendon reflexes are intact, no sensory abnormalities, normal pupillary light reflex

Myasthenia Gravis — Differential Diagnosis

- Eaton-Lambert Syndrome
  - *Increasing* muscle strength with repetitive movement
  - Associated with small cell carcinoma of the lung

Myasthenia Gravis — Diagnosis

- Best initial test
  - Acetylcholine receptor antibody test
  - Virtually diagnostic with generalized symptoms (80-90%)
  - Less diagnostic with disease limited to the eyes (70%)

- Edrophonium (Tensilon®) test
  - Sensitive but not specific

Myasthenia Gravis — Diagnosis

- Chest X-Ray
  - Rule out thymoma
- Most Accurate test
  - EMG (electromyelography)- decremental decrease in muscle fiber contraction on repetitive nerve stimulation

Myasthenia Gravis — Tensilon® Test

Note ptosis

After the administration of tensilon

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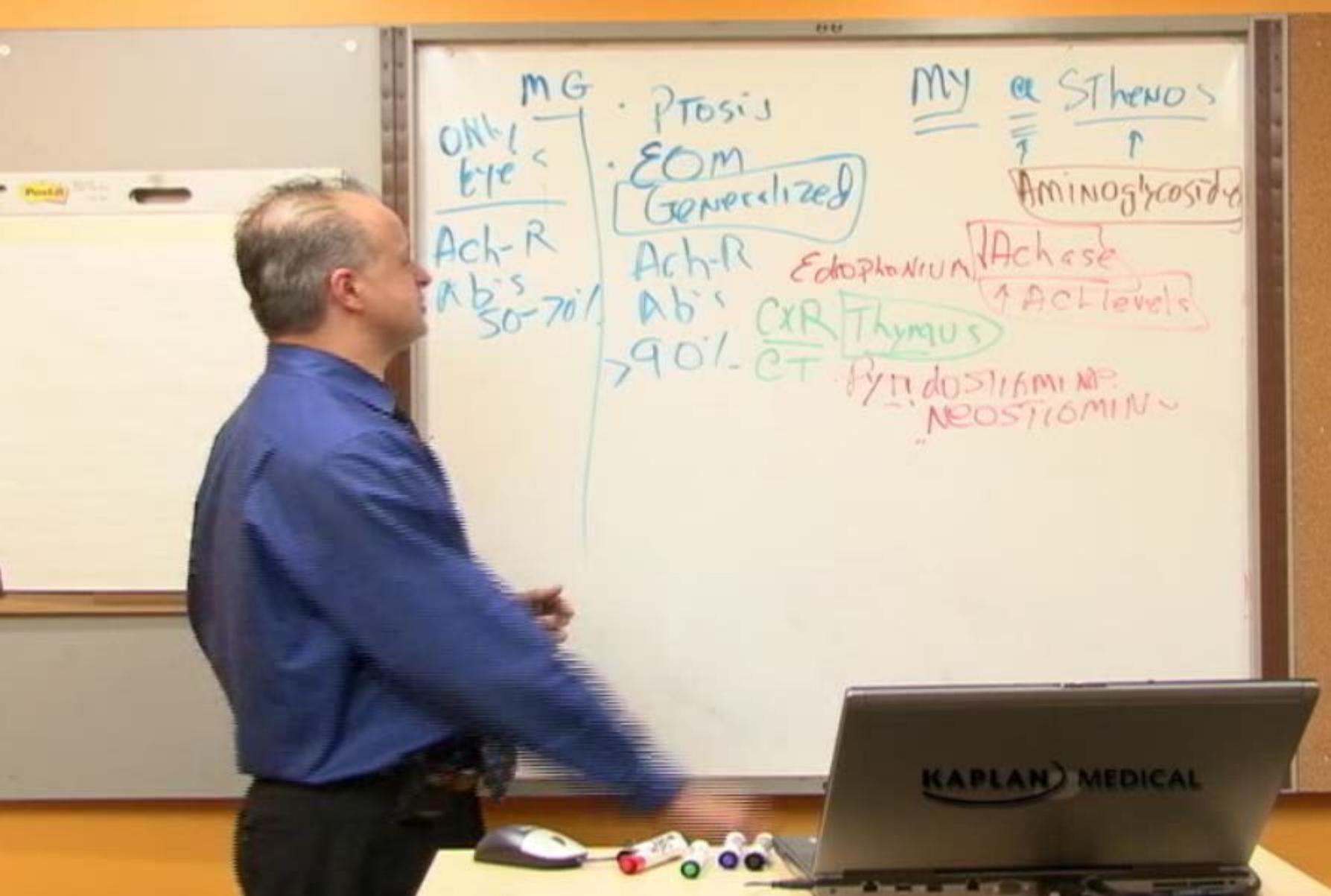
Myasthenia Gravis — Treatment

- Initial treatment for symptoms
  - Pyridostigmine or neostigmine
- If there is no response
  - Thymectomy
    - Post pubertal and < 60 years of age

Myasthenia Gravis — Treatment

- Still no response?
  - Immunosuppresive therapy
    - Steroids are the initial treatment of choice
    - If steroids fail- azathioprine usually added
    - Cyclosporine and cylophosphamide are alternatives (toxic)
    - If + respiratory symptoms: Plasmapheresis and intravenous immunoglobulin


MG
Ptosis
Only 1 eye
EOM
Generalized
Ach-R Abs 50-70%
Ach-R Abs >90%
CXR
Thymus
CT
MY
STHENOS
Aminoglycosides
Edrophonium
AChase
↑ ACh levels
Pyridostigmine
Neostigmine
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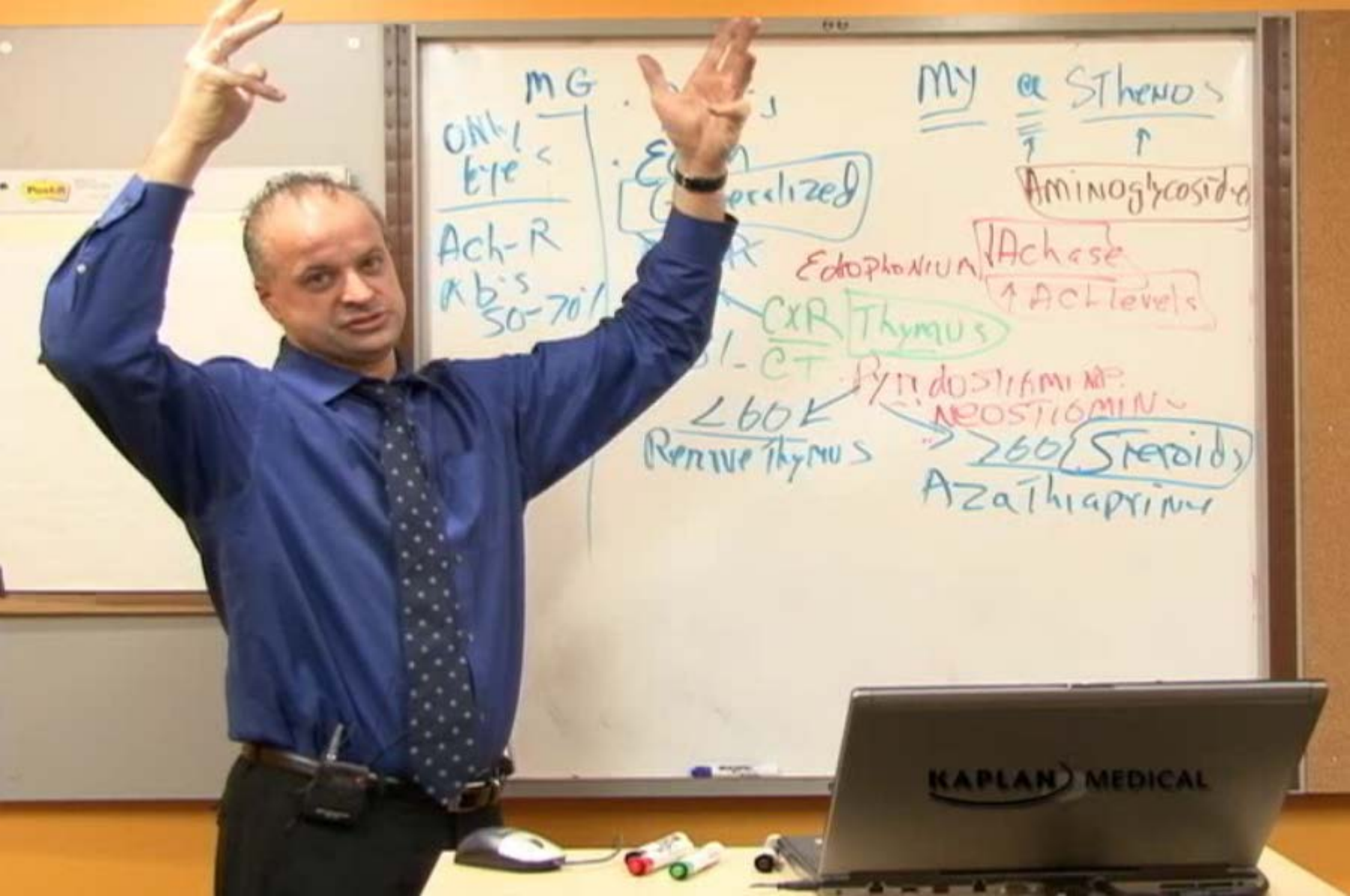
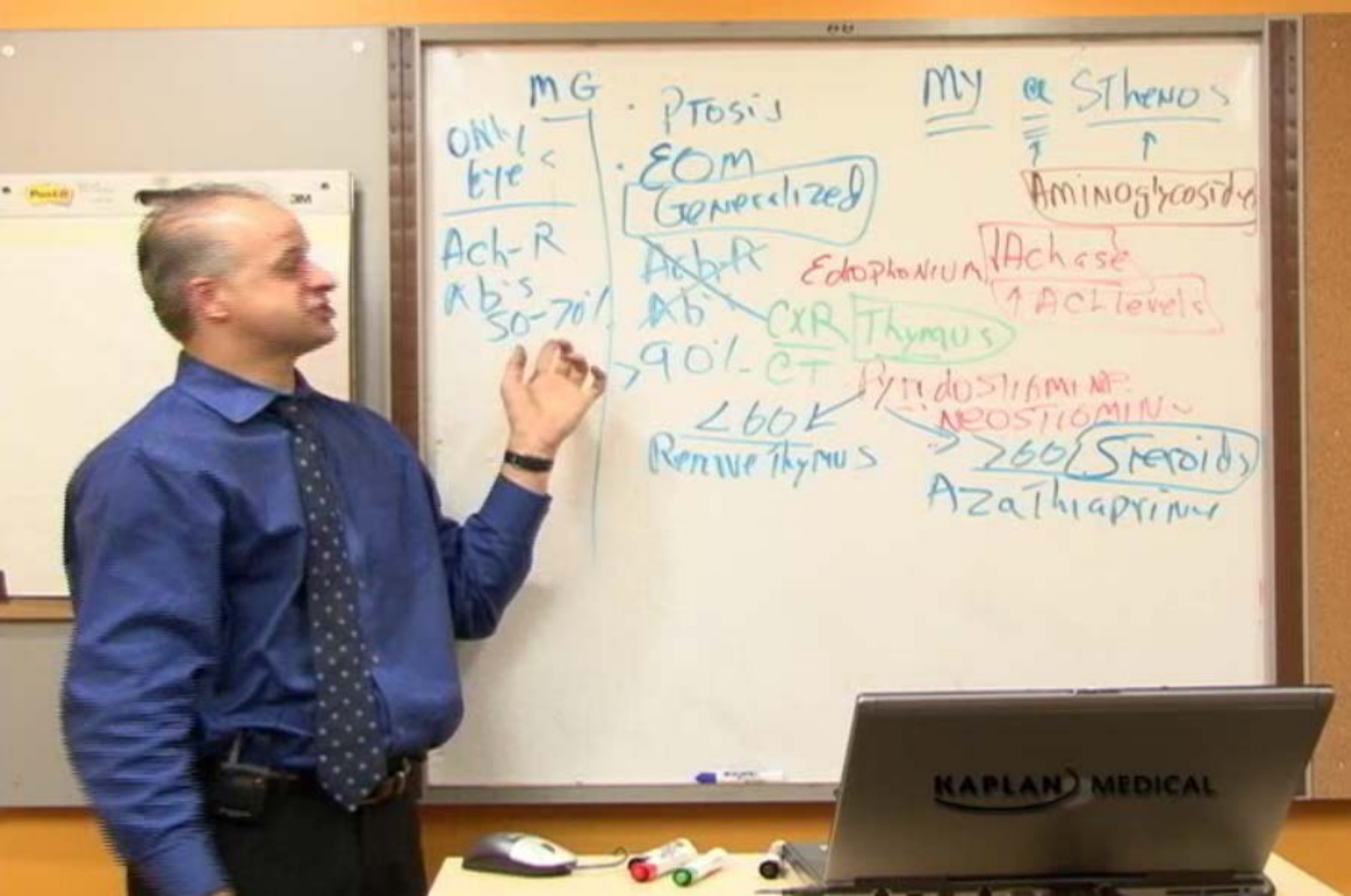

MG
Ach-R
Ab's
50-70%
Generalized
Edrophonium
AChase
↑ ACh levels
CXR
Thymus
Pyridostigmine
Neostigmine
< 60K
Remove Thymus
> 60
Steroids
Azathioprine
MY
STHENIA
↑
Aminoglycosides
KAPLAN MEDICAL


MG
Ptosis
EOM
Generalized
Ach-R
Abs
50-70%
>90%
CXR
Thymus
CT
<60K
Remove thymus
MY
STHENOS
Aminoglycosides
Edrophonium
AChase
↑ ACh levels
Pyridostigmine
Neostigmine
>60
Steroids
Azathioprine
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Ch. 6: Myasthenia Gravis

END

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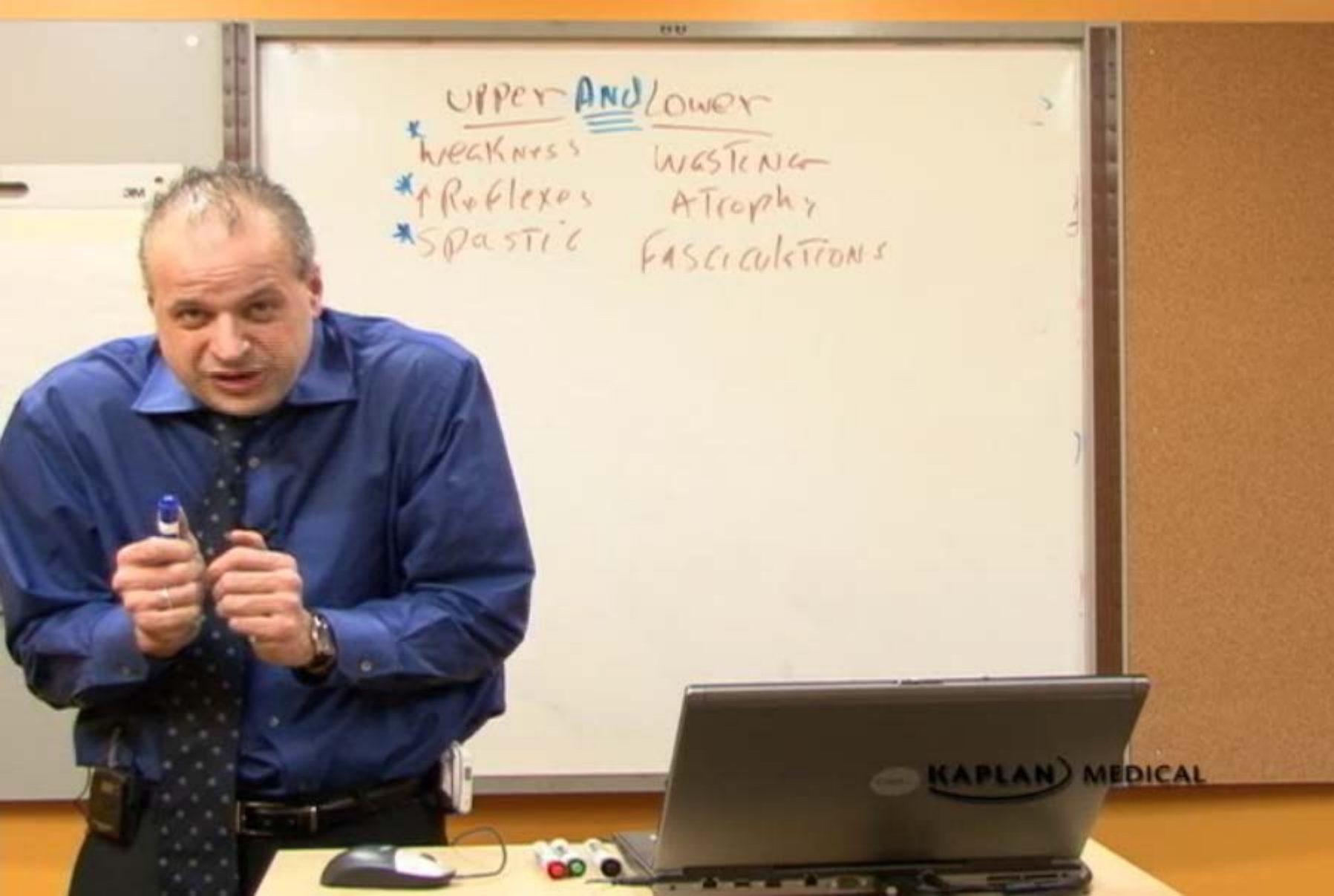
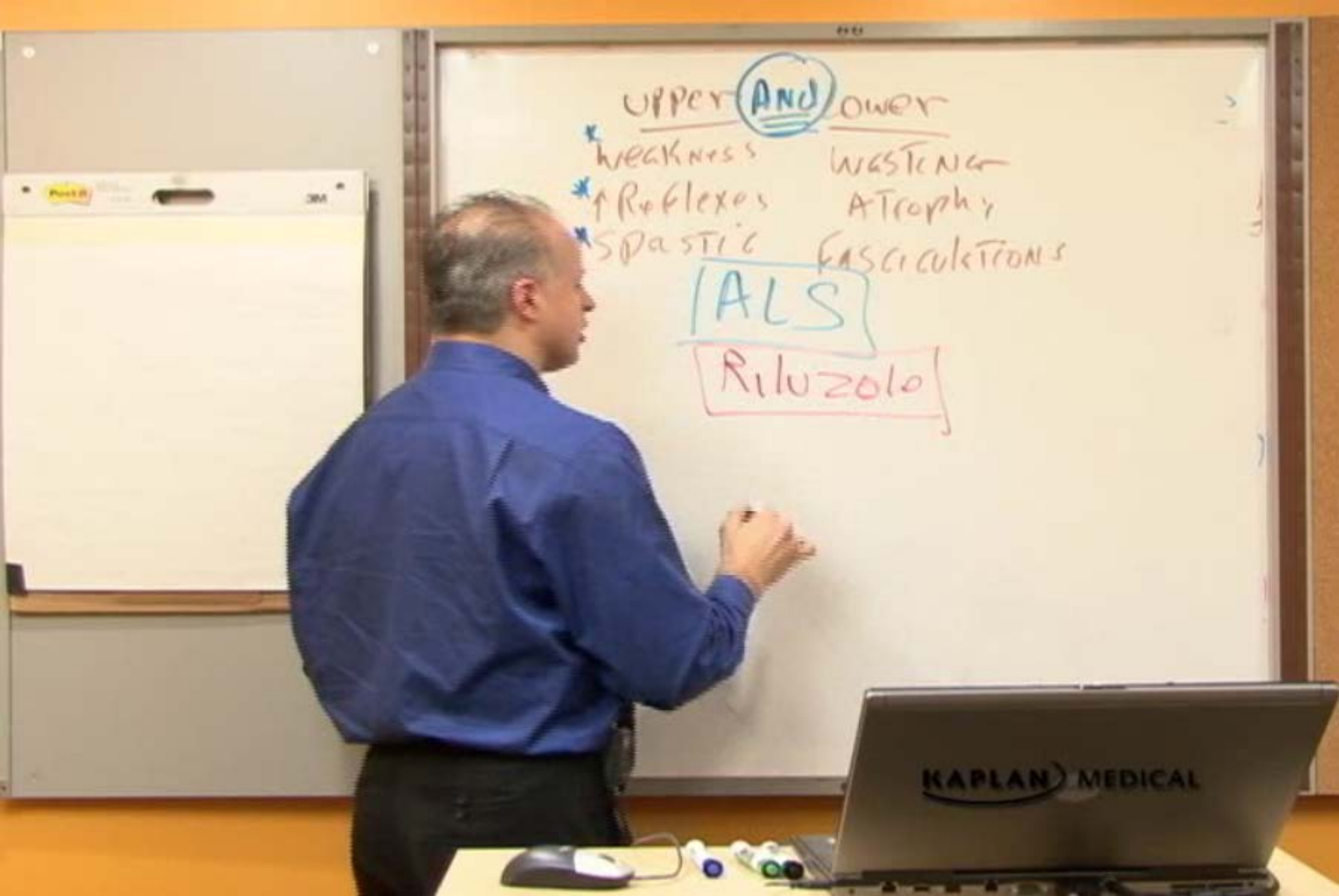
Ch. 7: Amyotrophic Lateral Sclerosis (ALS)


UPPER AND LOWER
WEAKNESS
↑ REFLEXES
SPASTIC
WASTING
ATROPHY
FASCICULATIONS
KAPLAN MEDICAL


UPPER AND LOWER
Weakness
↑ Reflexes
Spastic
Wasting
Atrophy
Fasciculations
ALS
Riluzole
KAPLAN MEDICAL

KAPLAN

MEDICAL

Ch. 7: Amyotrophic Lateral Sclerosis (ALS)

END

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MEDICAL

Ch. 8: Multiple Sclerosis

Multiple Sclerosis — Etiology

- Multifactorial
  - Genetic influence
  - Those above or below latitude 40 degrees North and 40 degrees South
    - Role for environmental trigger
      - » Infectious, dietary, climatic

Multiple Sclerosis — Clinical Presentation

Relapsing Remitting	Relapses of active disease with incomplete recovery between
Primary Progressive	Progressive from first onset with early disability
Secondary Progressive	Progressive with constant worsening of disease

Multiple Sclerosis — Diagnosis

- Clinical criteria
- Radiologic confirmation
  - MRI of the brain and spine most accurate
  - Increased T2 density and decreased T1 intensity in demyelinated plaques
  - Active MS lesions enhance with gadolinium and up to 6 weeks after

Multiple Sclerosis — Diagnosis

- Laboratory confirmation
  - CSF studies:
    - Mild pleocytosis, Mild elevation in total protein (levels >100 not MS)
  - If MRI of brain is negative, but suspicion is high
    - Check for oligoclonal bands in CSF

Multiple Sclerosis — Disease Modifying Therapy

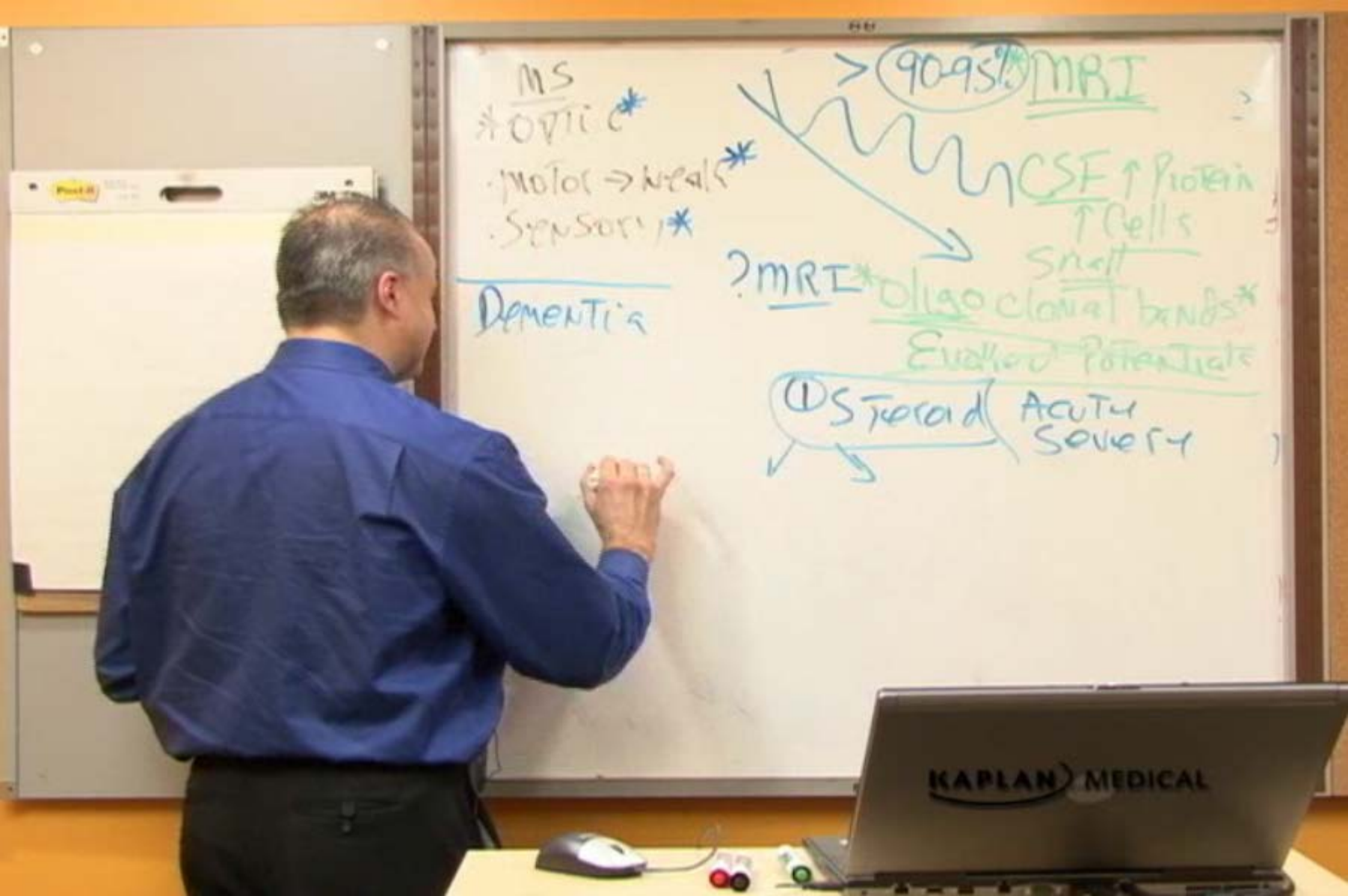
Relapsing Remitting	IFN-β1a IFN- β1b Glatiramer acetate (also known as copolymer I)
Primary Progressive	No approved therapy
Secondary Progressive	IFN- β1b Mitoxantrone

Multiple Sclerosis — Treatment of Acute Exacerbation

- Glucocorticoids x 3 days followed by 4 weeks per-oral taper
- Those unresponsive to steriods- plasma exchange

Multiple Sclerosis — Symptomatic Treatment

Spasticity	Baclofen (most effective) Tizandine and diazepam (nighttime)
Trigeminal neuralgia and dysesthesias	Carbamazepine, gabapentin, phenytoin, or TCAs
Bladder hyperreactivity	Oxybutynin
Urinary retention	Bethanechol
Fatigue	Amantidine or fluoxetine
Erectile dysfunction	Sildenafil


MS
Optic
Motor → Weak
Sensory
Dementia
> 90-95% MRI
CSF ↑ Protein
↑ Cells
Small
?MRI
Oligoclonal bands
Evoked Potentials
① Steroid
Acuity
Severity
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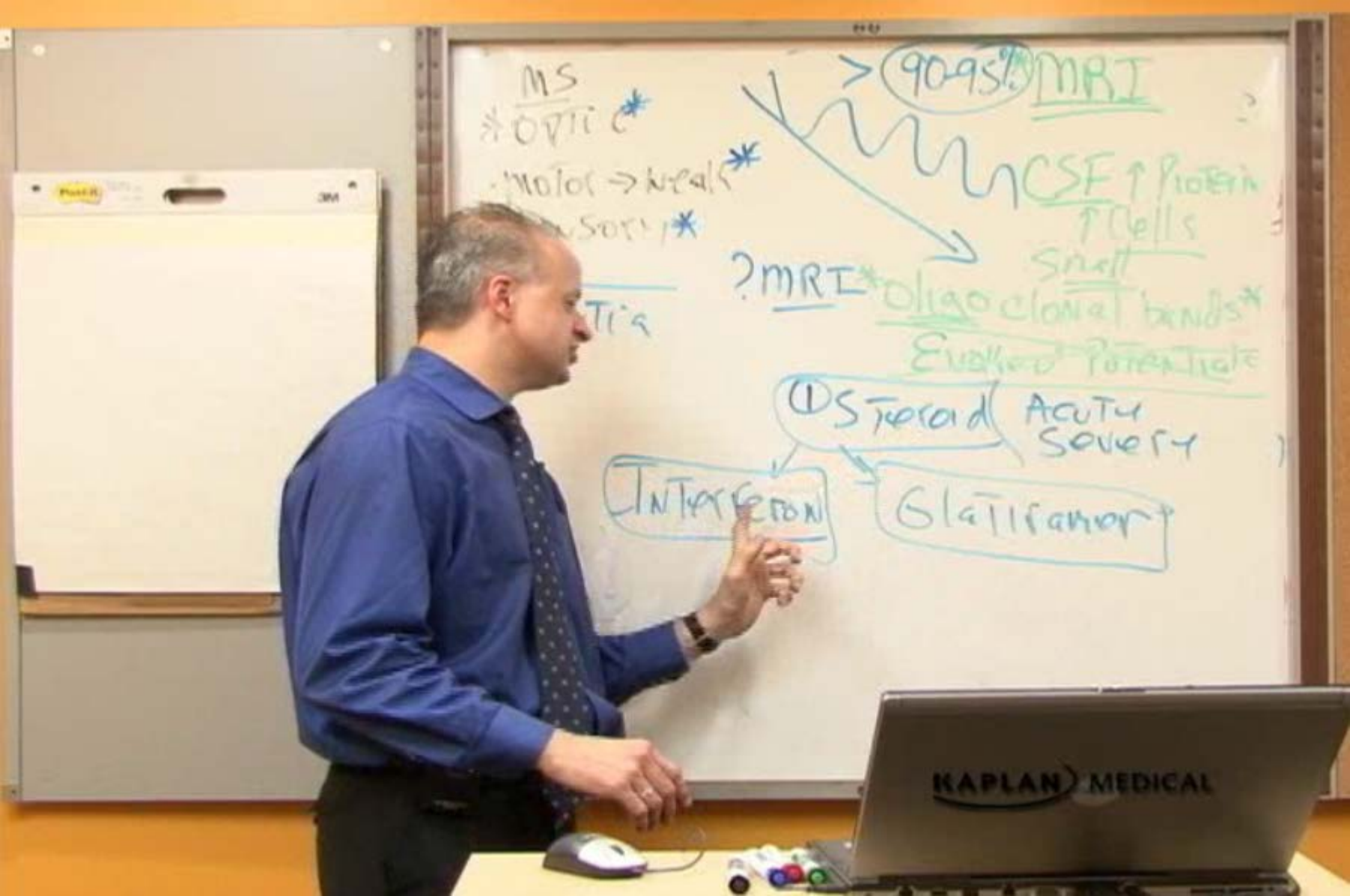
MS
optic
motor → weak
sory
?MRI
Ti's
> 90-95% MRI
CSF ↑ protein
↑ cells
small
oligoclonal bands
Evoked Potentials
① Steroid
Acute
Severe
Interferon
Glatiramer
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MEDICAL

Ch. 8: Multiple Sclerosis

END

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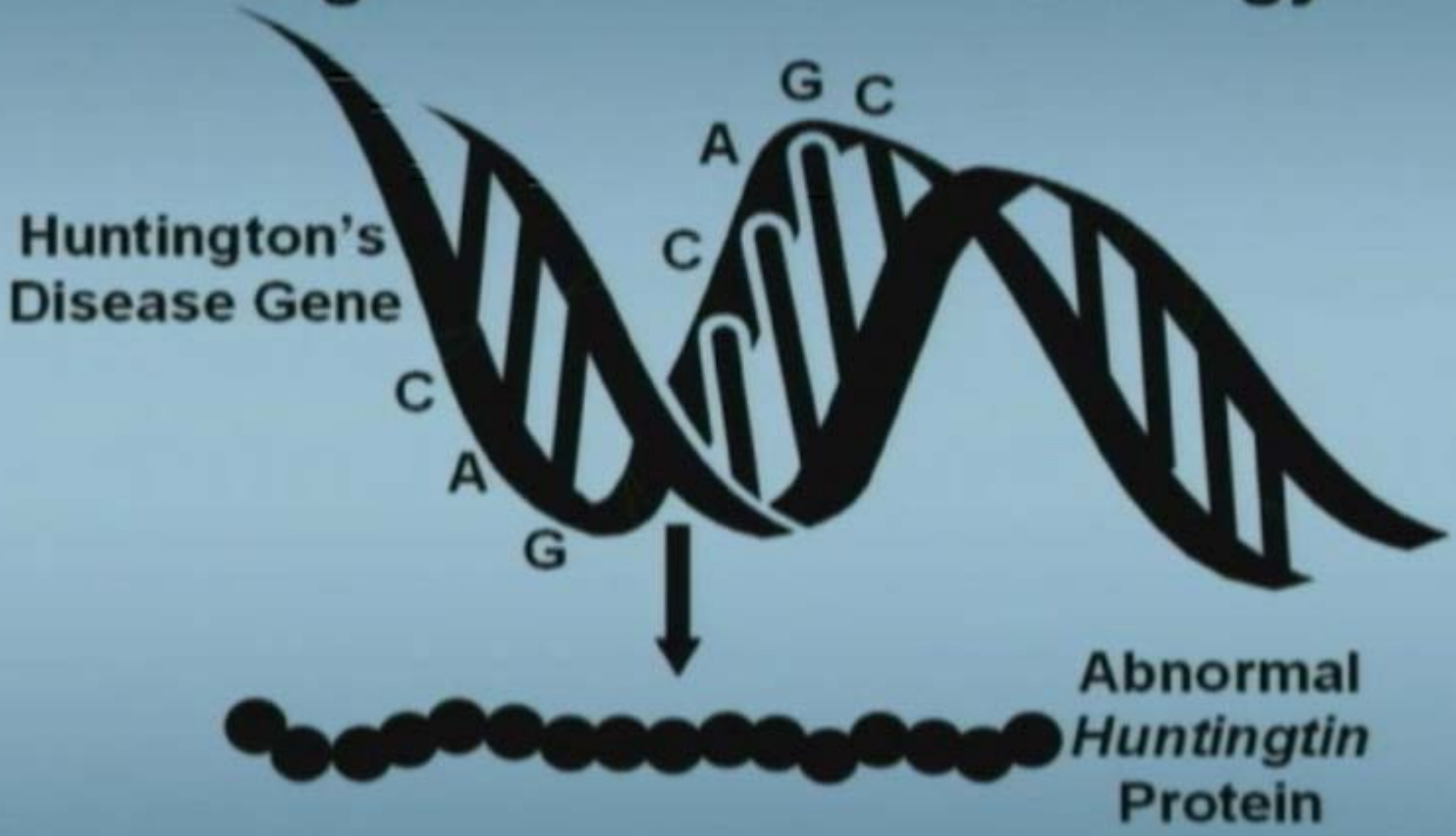
Dementia: Huntington's Disease

Huntington's Disease — Etiology

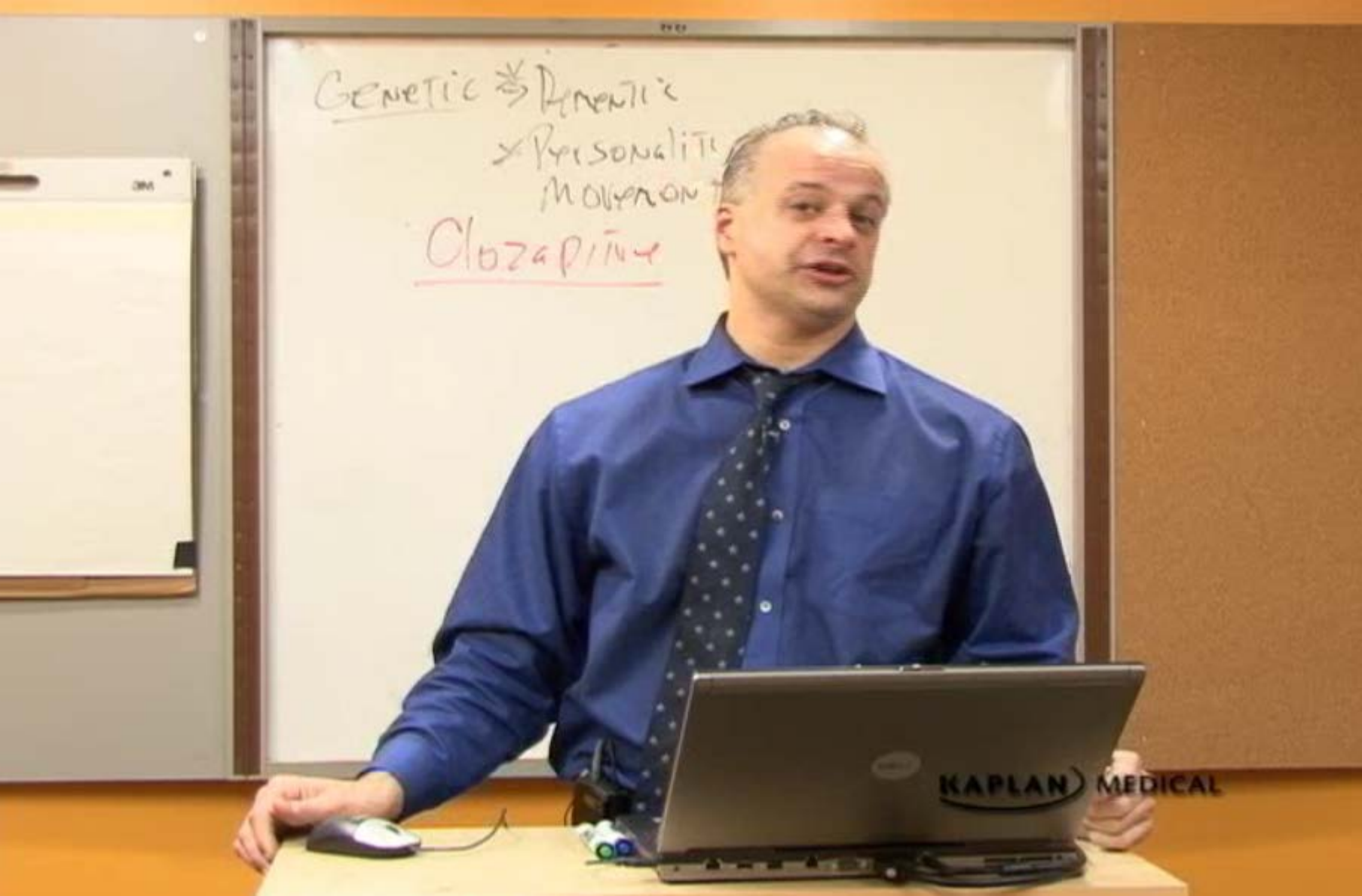
- Autosomal dominant
- Gene located on chromosome 4p codes for *Huntingtin* protein
- CAG trinucleotide repeat expansion
- Abnormal protein cleavage

Huntington's Disease — Etiology



Huntington's Disease — Diagnosis and Treatment

- Diagnosis
  - Genetic testing (DNA)
  - CT scans- cerebral atrophy, atrophy of the caudate late in disease
- Treatment
  - Clozapine for behavioral changes


Clozapine
KAPLAN MEDICAL

KAPLAN

MEDICAL

Dementia: Huntington's Disease

END

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Ch. 10: Parkinson's Disease and Other Movement Disorders

Parkinson's Disease — Syndromes

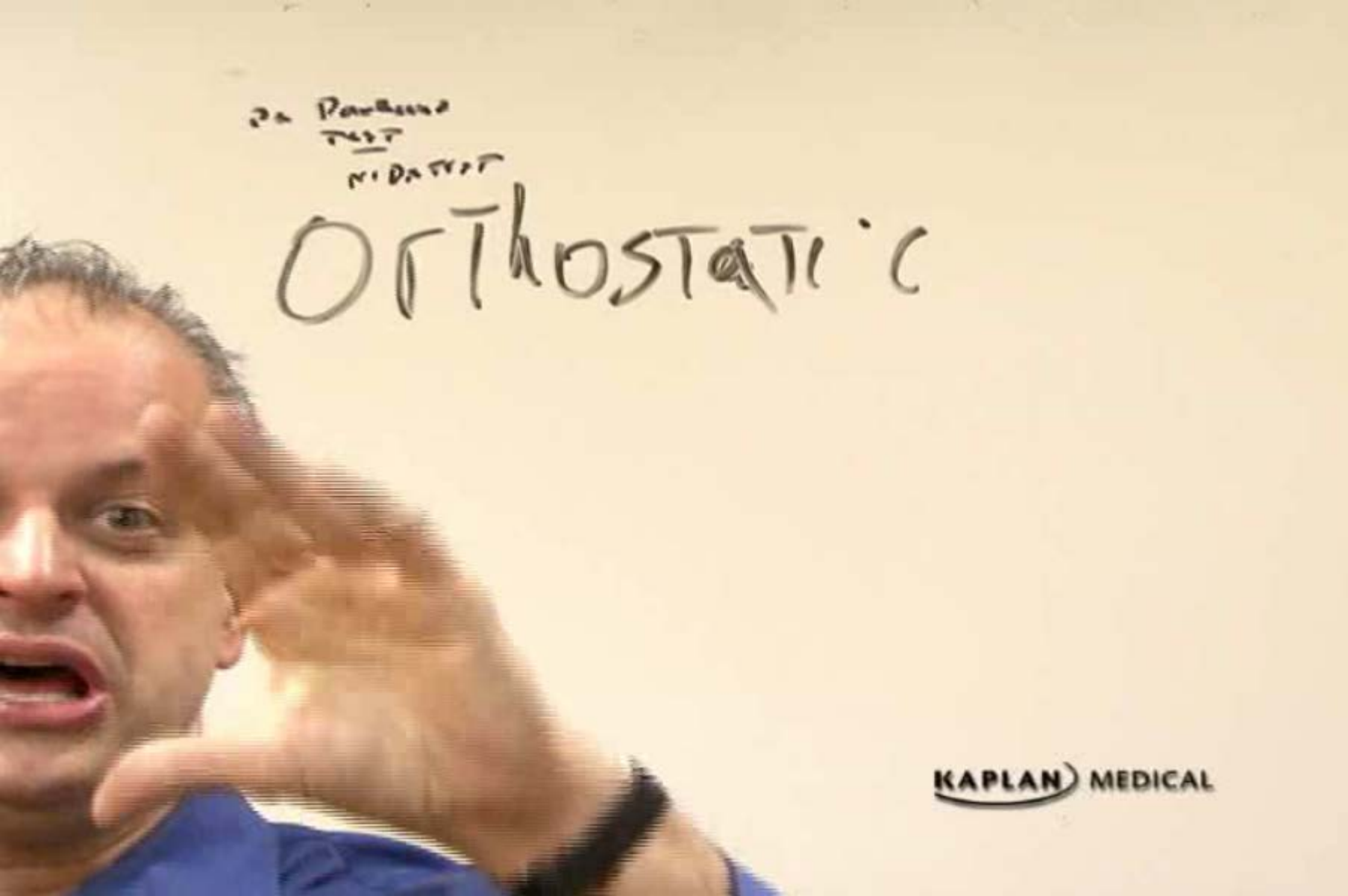
Supranuclear Palsy	Parkinsonism + vertical gaze palsy
Olivopontocerebellar Atrophy	Parkinsonism + prominent ataxia
Shy-Drager Syndrome	Parkinsonism + Prominent orthostatic hypotension

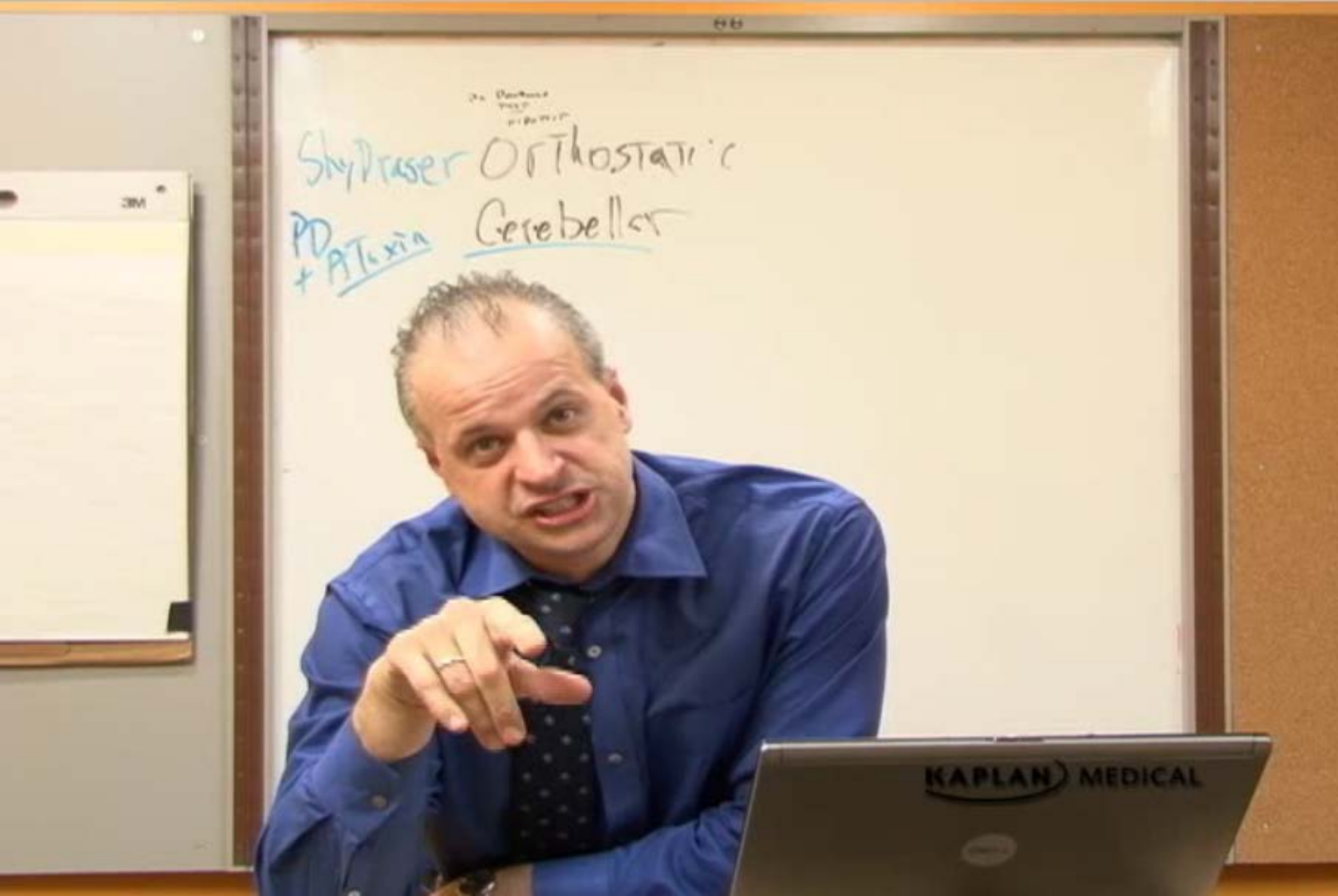
Parkinson's Disease — Treatment

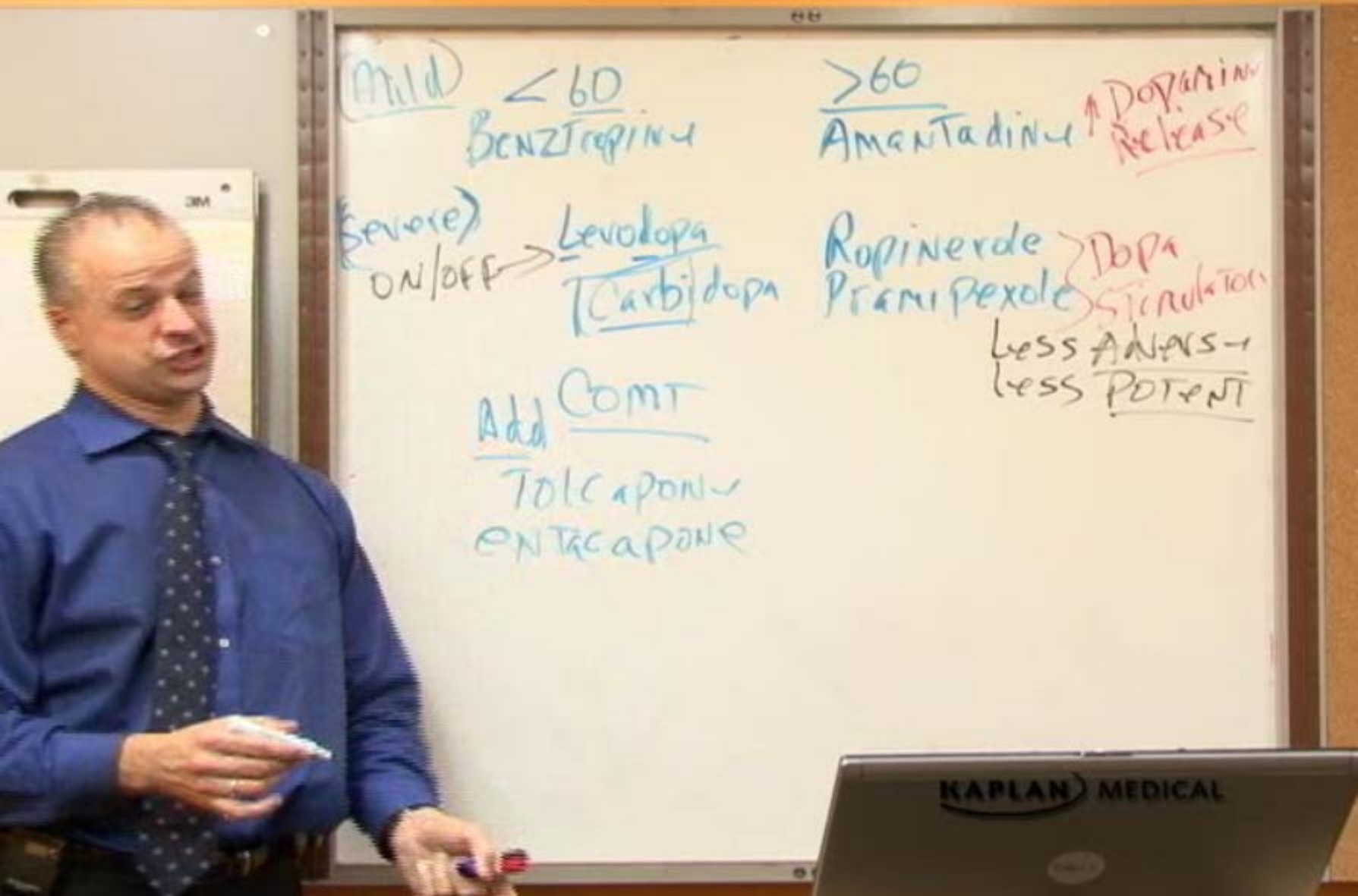
Dopamine agonists	Anticholinergics
• Carbidopa/ Levodopa •Pramiprexole, bromocriptine, pergolide and ropinirole (direct) •COMT inhibitors (tolcapone and entacapone), selegiline, amantadine (indirect)	• Benztropine • Trihexyphenidyl

Parkinson's Disease — Treatment

Patient Population	Drug of Choice
Functional status is intact and < 60 years of age	Start anti-cholinergic
Functional status is intact and > 60 years of age	Start amantidine
Compromised functional status	Start carbidopa/ levodopa


Orthostatic
KAPLAN MEDICAL


Shy Drager
Orthostatic
PD
+ Ataxia
Cerebellar
KAPLAN MEDICAL


Mild
< 60
Benztropine
> 60
Amantadine
↑ Dopamine Release
Severe
ON/OFF
Levodopa
Carbidopa
Ropinerole
Pramipexole
Dopa Stimulation
Less Advers-
less Potent
Add
COMT
Tolcapone
Entacapone
KAPLAN MEDICAL

KAPLAN

MEDICAL

Parkinson's Disease

END

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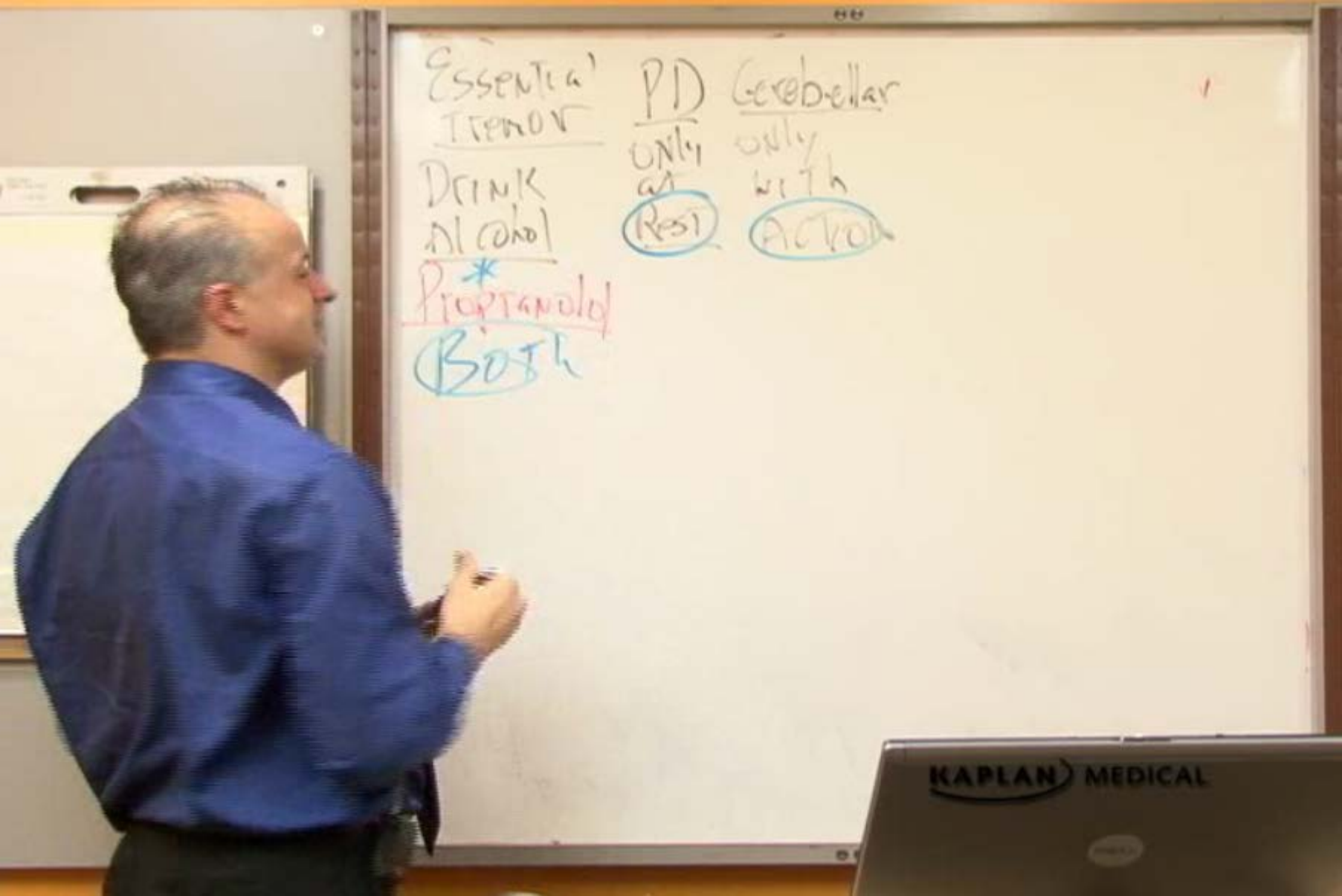
MEDICAL

Parkinson's: Benign Essential Tremor


Essential Tremor
PD
Cerebellar
Drink Alcohol
only at Rest
only with Action
*
Propranolol
Both
KAPLAN MEDICAL

KAPLAN

MEDICAL

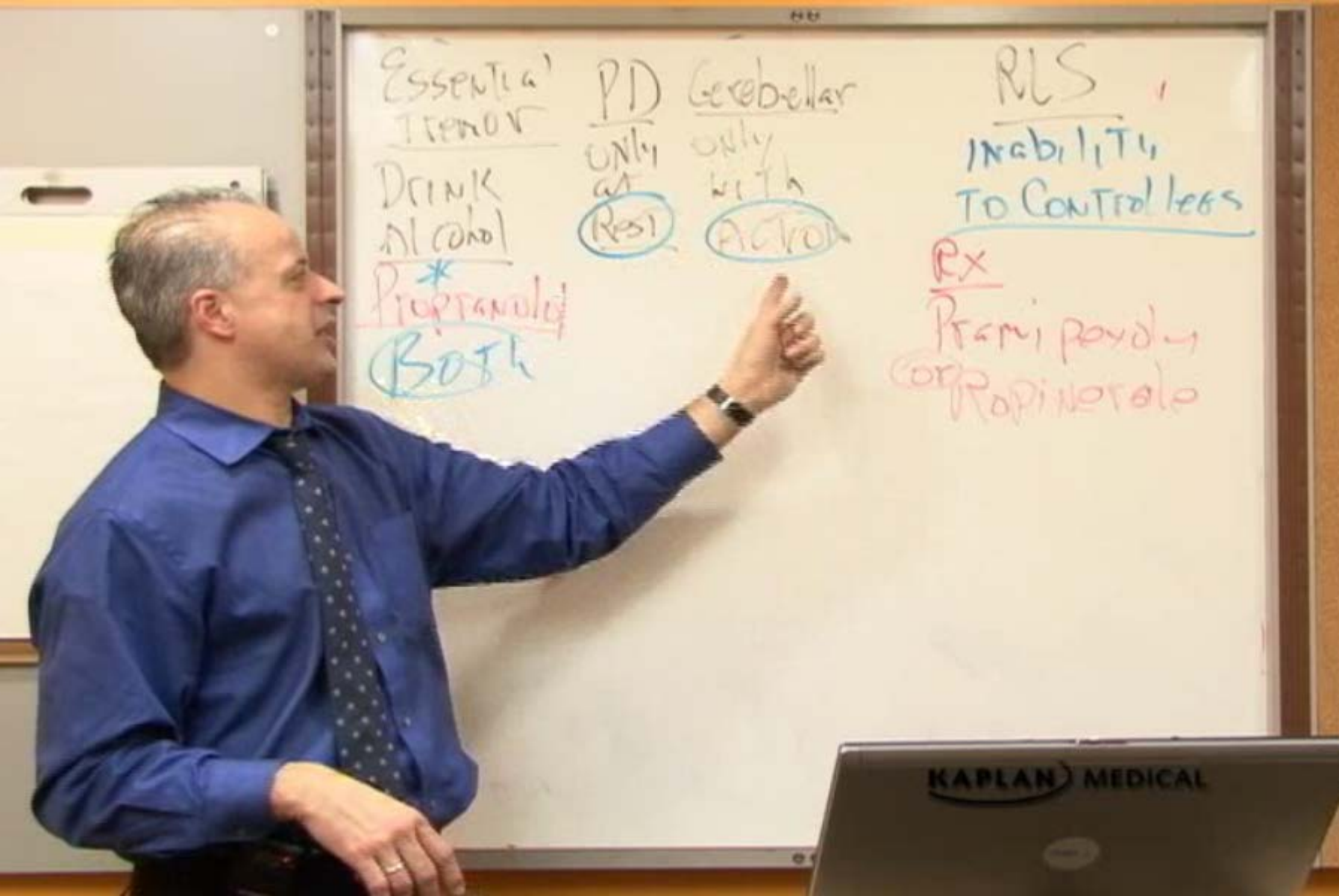
Parkinson's: Benign Essential Tremor

END

KAPLAN

MEDICAL

Parkinson's: Restless Leg Syndrome


Essential Tremor
Drink Alcohol
* Propranolol
Both
PD
only at Rest
Cerebellar
only with Action
RLS
Inability To Controll legs
Rx
Pramipexole
or Ropinerole
KAPLAN MEDICAL

KAPLAN

MEDICAL

Parkinson's: Restless Leg Syndrome

END